



Laura Kestilä

# **PATHWAYS TO HEALTH**

## **Determinants of Health, Health Behaviour and Health Inequalities in Early Adulthood**

Publications of the National Public Health Institute  23/2008

Department of Health and Functional Capacity  
National Public Health Institute, Helsinki, Finland  
*and*

Department of Sociology,  
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Helsinki, Finland 2008

**Laura Kestilä**

**PATHWAYS TO HEALTH**

DETERMINANTS OF HEALTH, HEALTH  
BEHAVIOUR AND HEALTH INEQUALITIES  
IN EARLY ADULTHOOD

ACADEMIC DISSERTATION

*To be presented with the permission of the Faculty of Social Sciences of the  
University of Helsinki, for public examination in the Auditorium of the Arppeanum,  
Snellmaninkatu 3, on October 24, 2008, at 12 noon.*

National Public Health Institute, Helsinki, Finland

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Department of Sociology,  
University of Helsinki, Finland

Helsinki 2008

## **Publications of the National Public Health Institute KTL A23 / 2008**

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### **Julkaisija-Utgivare-Publisher**

#### **Kansanterveyslaitos (KTL)**

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00300 Helsinki

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Telephone +358 9 474 41, telefax +358 9 4744 8408

ISBN 978-951-740-865-3

ISSN 0359-3584

ISBN 978-951-740-866-0 (pdf)

ISSN 1458-6290 (pdf)

### **Kannen kuva - cover graphic: Jussi Eskola**

Yliopistopaino

Helsinki 2008

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**PATHWAYS TO HEALTH. Determinants of health, health behaviour and health inequalities in early adulthood.**

Publications of the National Public Health Institute, A23/2008, 164 Pages

ISBN 978-951-740-865-3; 978-951-740-866-0 (pdf version)

ISSN 0359-3584; 1458-6290 (pdf version)

<http://www.ktl.fi/portal/4043>

## **ABSTRACT**

There is increasing evidence that the origins of poor adult health and health inequalities can be traced back to circumstances preceding current socioeconomic position and living conditions. The life-course approach to examining the determinants of health has emphasised that exposure to adverse social and economic circumstances in earlier life or concurrent adverse circumstances due to unfavourable living conditions in earlier life may lead to poor health, health-damaging behaviour, disease or even premature death in adulthood.

There is, however, still a lack of knowledge about the contribution of social and economic circumstances in childhood and youth to adult health and health inequalities, and even less is known about how environmental and behavioural factors in adulthood mediate the effects of earlier adverse experiences. The main purpose of this study was to deepen our understanding of the development of poor health, health-damaging behaviours and health inequalities during the life-course. Its aim was to find out which factors in earlier and current circumstances determine health, the most detrimental indicators of health behaviour (smoking, heavy drinking and obesity as a proxy for the balance between nutrition and exercise), and educational health differences in young adults in Finland. Following the ideas of the social pathway theory, it was assumed that childhood environment affects adult health and its proximal determinants via different pathways, including educational, work and family careers. Early adulthood was studied as a significant phase of life when many behavioural patterns and living conditions relevant to health are established. In addition, socioeconomic health inequalities seem to emerge rapidly when moving into adulthood; they are very small or non-existent in childhood and adolescence, but very marked by early middle age.

The data of this study were collected in 2000–2001 as part of the Health 2000 Survey (N=9,922), a cross-sectional and nationally representative health interview and examination survey. The main subset of data used in this thesis was the one comprising the age group 18–29 years (N=1,894), which included information collected by standardised structured computer-aided interviews and self-administered questionnaires. The survey had a very high participation rate at almost 90% for the core questions.

According to the results of this study, childhood circumstances predict the health of young adults. Almost all the childhood adversities studied were found to be associated

with poor self-rated health and psychological distress in early adulthood, although fewer associations were found with the somatic morbidity typical of young adults. These effects seemed to be more or less independent of the young adult's own education. Childhood circumstances also had a strong effect on smoking and heavy drinking, although current circumstances, and education in particular, played a role in mediating this effect. Parental smoking and alcohol abuse had an influence on the corresponding behaviours of offspring. Childhood circumstances had a role in the development of obesity and, to a lesser extent, overweight, particularly in women. The findings support the notion that parental education has a strong effect on early adult obesity, even independently of the young adult's own educational level.

There were marked educational differences in self-rated health in early adulthood: those in the lowest educational category were most likely to have average or poorer health. Childhood social circumstances seemed to explain a substantial part of these educational differences. In addition, daily smoking and heavy drinking contributed substantially to educational health differences. However, the contribution of childhood circumstances was largely shared with health behaviours adopted by early adulthood. Employment also shared the effects of childhood circumstances on educational health differences.

The results indicate that childhood circumstances are important in determining health, health behaviour and health inequalities in early adulthood. Early recognition of childhood adversities followed by relevant support measures may play an important role in preventing the unfortunate pathways leading to the development of poor health, health-damaging behaviour and health inequalities. It is crucially important to recognise the needs of children living in adverse circumstances as well as children of substance abusing parents. In addition, single-parent families would benefit from support.

Differences in health and health behaviours between different sub-groups of the population mean that we can expect to see ever greater health differences when today's generation of young adults grows older. This presents a formidable challenge to national health and social policy as well as health promotion. Young adults with no more than primary level education are at greatest risk of poor health. Preventive policies should emphasise the role of low educational level as a key determinant of health-damaging behaviours and poor health.

**Keywords:** health, health behaviour, health inequalities, life-course, socioeconomic position, education, childhood circumstances, self-rated health, psychological distress, somatic morbidity, smoking, heavy drinking, BMI, early adulthood

**Laura Kestilä**

**POLUT TERVEYTEEN. Nuorten aikuisten terveyden, terveyskäyttäytymisen ja terveyserojen määrittäjät.**

Kansanterveyslaitoksen julkaisuja, A23/2008, 164 sivua

ISBN 978-951-740-865-3; 978-951-740-866-0 (pdf-versio)

ISSN 0359-3584; 1458-6290 (pdf-versio)

<http://www.ktl.fi/portal/4043>

## **TIIVISTELMÄ**

Aikaisemmat tutkimukset viittaavat yhä vahvemmin siihen, että aikuisiän terveys ja terveyskäyttäytyminen juontavat juurensa nykyistä sosiaalista asemaa ja elinoloja edeltävistä elämänvaiheista. Elämänkulkunäkökulma terveyden määrittäjien tutkimuksessa korostaa mm. sitä, että huonoille sosiaalisille ja taloudellisille elinoloille altistuminen lapsuudessa, tai niistä seuranneet epäsuotuisat elinolot aikuisuudessa, voivat johtaa huonoon terveyteen, terveyttä vaarantavaan käyttäytymiseen, sairauteen tai jopa ennenaikaiseen kuolemaan aikuisuudessa.

Vielä ei tiedetä kuitenkaan riittävästi siitä, miten lapsuuden ja nuoruuden elinolot vaikuttavat aikuisuuden terveyteen ja terveyseroihin. Vielä vähemmän tiedetään siitä, miten myöhempien elämänvaiheiden elinolot ja terveyskäyttäytymiseen liittyvät piirteet välittävät aikaisempien elinolojen vaikutuksia. Tämän väitöskirjan tarkoituksena on ollut syventää ymmärrystä terveyden, terveyskäyttäytymisen ja terveyserojen kehittämisestä elämänkulussa. Tavoitteena on ollut selvittää, miten aikaisemmat ja nykyiset elinolot määrittävät terveyttä, keskeisimpiä haitallisen terveyskäyttäytymisen muotoja sekä koulutusryhmien välisiä terveyseroja suomalaisilla nuorilla aikuisilla. Sosiaalisten polkujen teoriaa mukaillen oletettiin, että lapsuuden elinympäristö määrittää nuorten aikuisten terveyttä ja sen todennäköisiä määrittäjiä erilaisia väyliä pitkin, mm. koulutus-, työ- ja perheellistymisen polkujen kautta. Nuori aikuisuus on merkittävänä elämänvaiheena tutkimuksen kohteena, sillä silloin monet myöhemmän terveyden kannalta olennaiset terveyskäyttäytymisen muodot ja elinolot vakiintuvat. Lisäksi sosioekonomisten terveyserojen on todettu ilmaantuvan nopeasti aikuisuuden kynnyksellä ja olevan suuria jo varhaisessa keski-ikässä.

Tutkimuksen aineisto on kerätty vuosina 2000–2001 osana Terveys 2000 -tutkimusta (N=9 922), joka oli koko maata edustava terveyshaastatteluihin ja terveystarkastuksiin perustuva tutkimus. Tässä väitöskirjassa käytettiin pääosin tutkimuksen 18–29-vuotiaita nuoria aikuisia (N=1 894) edustavaa otosta, josta koottiin tietoa terveyshaastattelun ja kyselyn avulla. Tutkimuksen osallistumisprosentti oli korkea (lähes 90 % sen ydinkysymyksiin).

Tulosten mukaan lapsuuden epäsuotuisat elinolot ennustavat nuoren aikuisiän huonoa terveyttä. Monien lapsuuden sosiaalisten ongelmien havaittiin olevan yhteydessä nuorten aikuisten huonoon koettuun terveyteen ja psyykkiseen kuormittuneisuuteen,

mutta yhteydet nuorille aikuisille tyypilliseen somaattiseen sairastavuuteen olivat vähäisempiä. Lapsuuden elinolojen vaikutukset terveyteen näyttivät olevan melko riippumattomia nuoren aikuisen omasta koulutuksesta. Lapsuuden elinolot ennustivat voimakkaasti myös nuorten aikuisten päivittäistä tupakointia ja alkoholin suurkulutusta, ja nykyiset elinolot, erityisesti vastaajan oma koulutus, näyttivät välittävän osin näitä vaikutuksia. Erityisesti vanhempien tupakointi ja alkoholinkäyttö ennustivat heidän jälkeläistensä vastaavia terveyskäyttäytymisen muotoja. Lapsuuden elinolot vaikuttivat myös ylipainon ja etenkin lihavuuden kehittymiseen, erityisesti naisilla. Varsinkin vanhempien vähäisellä koulutuksella oli voimakas yhteys nuoren aikuisen lihavuuteen.

Nuorilla aikuisilla havaittiin selviä koulutusryhmien välisiä eroja koetussa terveydessä. Alimpaan koulutusluokkaan kuuluvista keskimääräistä selvästi suurempi osa ilmoitti terveytensä olevan keskitasoinen tai sitä huonompi. Tulosten mukaan lapsuuden elinolot ja ongelmat selittävät koulutusryhmien välisistä terveyseroista selvän osan. Lisäksi päivittäisellä tupakoinnilla ja alkoholin suurkulutuksella näytti olevan suuri selittävä vaikutus. Lapsuuden elinolojen vaikutus näyttää liittyvän kuitenkin selvästi nuoreen aikuisuuteen mennessä omaksuttuun terveyskäyttäytymiseen. Myös työllistyminen näyttää jakavan lapsuuden elinolojen vaikutuksia koulutusryhmien välisiin terveyseroihin.

Tulokset osoittavat, että lapsuuden elinolot ovat tärkeitä terveyden, terveyskäyttäytymisen ja terveyserojen määrittäjiä nuorena aikuisuudessa. Lapsuuden epäsuotuisien olosuhteiden ja ongelmien varhainen tunnistaminen sekä niiden pohjalta kehitetyt tukitoimet, voivat osaltaan ehkäistä sellaisten epäsuotuisien polkujen synnyn, jotka johtavat huonoon terveyteen, haitalliseen terveyskäyttäytymiseen ja väestöryhmien välisiin terveyseroihin. Ongelmallisissa elinoloissa sekä päihteitä käyttävissä perheissä elävien lasten tarpeiden tunnistaminen olisi tärkeää. Lisäksi yksinhuoltajaperheiden tilanteisiin tulisi kiinnittää erityistä huomiota.

Nuorten aikuisten terveyden tulevaisuuden näkymät asettavat monia haasteita kansalliselle terveys- ja sosiaalipolitiikalle sekä terveyden edistämiselle. Tutkimuksessa havaitut selvät väestöryhmien väliset erot terveydessä ja terveyskäyttäytymisessä ennustavat jyrkkiä terveydentilan ja hyvinvoinnin eroja nuorten aikuisten sukupolven varttuessa. Nuoret aikuiset, joilla on vain perusasteen tutkinto ovat suurimmassa vaarassa tulevaisuuden terveyden kannalta. Alhaisen koulutuksen suuri merkitys huonon terveyden ja haitallisen terveyskäyttäytymisen riskitekijänä tulisi ottaa huomioon suunniteltaessa ehkäiseviä ja terveyttä edistäviä toimenpiteitä.

**Asiasanat:** terveys, terveyskäyttäytyminen, terveyserot, elämäntapa, sosioekonominen asema, koulutus, lapsuuden elinolot, koettu terveys, psyykinen kuormittuneisuus, somaattinen sairastavuus, tupakointi, alkoholin suurkulutus, BMI, nuori aikuisuus



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## ABBREVIATIONS

ACE	adverse childhood experiences
ACE Study	Adverse Childhood Experiences -study, United States
BMI	body mass index
CAPI	computer-aided personal interview
CCA	cumulative childhood adversities
CI	confidence interval
CHD	coronary heart disease
CHI2	chi-square test for significance of difference
GHQ	General Health Questionnaire
GHQ12	12-item version of the General Health Questionnaire
HeSSup	Sosiaalisen tuen terveystaikutukset -seurantatutkimus [Health and Social Support in Finland – follow-up study]
HHS	Helsinki Health Study, Finland
ICD	International Classification of Diseases
N	number
OR	odds ratio
OECD	Organisation for Economic Co-operation and Development
r	Pearson correlation coefficient
RRR	relative risk ratio
SEP	socioeconomic position
SRH	self-rated health
TAM	Tamperelaisnuorten Mielenterveys -seurantatutkimus [Stress development and mental health. A prospective follow-up study of adolescents], Finland
WHO	World Health Organization

## LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original articles referred to in the text by their Roman numerals:

- I      Kestilä L, Koskinen S, Martelin T, Rahkonen O, Pensola T, Aro H and Aromaa A. Determinants of health in early adulthood: what is the role of parental education, childhood adversities and own education?  
*Eur J Public Health*. 2006 Jun; 16(3):306–15.
- II     Kestilä L, Koskinen S, Martelin T, Rahkonen O, Pensola T, Pirkola S, Patja K and Aromaa A. Influence of parental education, childhood adversities, and current living conditions on daily smoking in early adulthood.  
*Eur J Public Health*. 2006 Dec; 16(6):617–26.
- III    Kestilä L, Martelin T, Rahkonen O, Joutsenniemi K, Pirkola S, Poikolainen K and Koskinen S. Childhood and current determinants of heavy drinking in early adulthood.  
*Alcohol and Alcoholism* 2008 Jul-Aug; 43(4):460–9.
- IV    Kestilä L, Rahkonen O, Martelin T, Lahti-Koski M and Koskinen S. Do childhood social circumstances affect overweight and obesity in early adulthood? *Scandinavian Journal of Public Health*, in press.
- V      Kestilä L, Martelin T, Rahkonen O, Härkänen T and Koskinen S. The contribution of childhood circumstances, current circumstances and health behaviour to educational health differences in early adulthood.  
*Submitted*.

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# 1 INTRODUCTION

The origins of poor adult health and health inequalities can be traced back to the circumstances preceding current socioeconomic position (SEP) and living conditions. The life-course approach to the epidemiology of health and health inequalities emphasises that the biological and social beginnings of life are crucial to the individual's potential for adult health (Power and Hertzman 1997; Kuh and Hardy 2002; Davey Smith 2003; Kuh and Ben-Shlomo 2004). Regarding social pathways to health, it has been reported that long-term exposure to physical risks or adverse social and economic circumstances in childhood (Wadsworth 1997; Hertzman, Power et al. 2001) or concurrent adverse circumstances due to unfavourable circumstances in earlier life may lead to poor health, health-damaging behaviour, disease or even premature death in adulthood. For example, the effects of economic and social hardship in childhood on subsequent adult health may be partly mediated through youth paths, education and employment, or through other adult circumstances (see e.g. Lundberg 1993; Lundberg 1997; Pensola and Martikainen 2003; Pensola 2004; Mäkinen, Laaksonen et al. 2006). In addition, health may be determined by early life influences together with adult experiences. The association between exposure and health outcome may be mediated by a risk or protective factor when it chronologically follows the exposure and is conceptualised as lying, at least partly, on the causal pathway (Kuh, Ben-Shlomo et al. 2003). Perhaps the most frequently hypothesised pathway – or mediating variable – between primary circumstances and adult health is adult SEP. Adult SEP is considered a pathway, first of all because it is heavily influenced by primary SEP (Power and Matthews 1997), but it is itself predictive of many subsequent health outcomes. However, adult SEP is one important, but not the only, pathway linking primary SEP to adult health outcomes; employment paths and family formation, for example, may well play roles in the process as well.

The impacts of childhood living conditions and adversities on adult health are well-documented for several measures of health, including self-rated health and chronic diseases (Rahkonen, Lahelma et al. 1997; Dube, Felitti et al. 2003; Dong, Giles et al. 2004), psychological health (Sadowski, Ugarte et al. 1999; Levitan, Rector et al. 2003; Korkeila, Korkeila et al. 2005; Schilling, Aseltine et al. 2007) as well as mortality (Lynch, Kaplan et al. 1994; Davey Smith, Hart et al. 1998; Claussen, Davey Smith et al. 2003; Pensola 2004). In general, persons who have lived in poor economic and social childhood conditions tend to have poorer health in adulthood. In addition, it has been found that health-damaging behaviours in adulthood (such as smoking, excess alcohol use, physical inactivity and overweight) are connected with the primary social environment (Hope, Power et al. 1998; Parsons, Power et al. 1999; Anda, Whitfield et al. 2002; Huurre, Aro et al. 2003; Power, Graham et al. 2005). However, there is still a lot to know on the contribution of circumstances in childhood and youth to adult

health, and even less is known about how environmental and behavioural factors in adulthood mediate the effects of earlier adverse experiences. There is some evidence that primary SEP and childhood circumstances are associated with youth paths, such as tracks of education, employment and family formation, as well as later SEP (van de Mheen, Stronks et al. 1997; Pensola and Martikainen 2004). Various other assumptions have also been presented of potential explanatory pathways between primary SEP and adult health indicators. These causal mechanisms between adverse childhood experiences and adult health include physiological risk factors (Forsdahl 1978; Blane, Hart et al. 1996), lifestyle factors, such as smoking and physical activity (Blane, Hart et al. 1996; Lynch, Kaplan et al. 1997; van de Mheen, Stronks et al. 1998) as well as psychological and psychosocial mechanisms (Bosma, van de Mheen et al. 1999).

The foundations of health inequalities in adulthood are built from environmental and behavioural elements at different stages of the life-course. Various models based on theories of causation and selection have been developed in order to explain socioeconomic differences in health. Health differences according to SEP are generated by various factors and mechanisms, including material (structural), behavioural and psychosocial factors (van Oort, van Lenthe et al. 2005). Higher SEP may promote better living and healthier working conditions (Schrijvers, van de Mheen et al. 1998; Borg and Kristensen 2000; Monden 2005), as well as healthier lifestyles, attitudes and choices (Wardle and Steptoe 2003), and it is usually associated with physically less strenuous and psychosocially more rewarding work and better housing conditions than lower SEP. Moreover, compared with low-SEP persons, those with high SEP tend to have less health-damaging behaviours: they tend to smoke less (Paavola, Vartiainen et al. 2004; Laaksonen, Rahkonen et al. 2005; Power, Graham et al. 2005), drink less alcohol (Droomers, Schrijvers et al. 1999; Casswell, Pledger et al. 2003), be physically more active (Lindström, Hanson et al. 2001; Martinez-Gonzalez, Varo et al. 2001), have healthier nutrition habits (Roos, Talala et al. 2008) and are less likely to be obese (Sobal and Stunkard 1989). Health and health behaviour in childhood and adolescence may also have influence on adult SEP; those with poorer health (Haas 2006) and health-damaging lifestyles (Koivusilta, Rimpelä et al. 1998) may end up in lower socioeconomic destinations in adulthood.

Health inequalities may arise from circumstances and experiences in childhood which affect one's education, employment, living conditions and health behaviour and further, health. Childhood environment can explain socioeconomic health differences if it is associated with both adult SEP and health in adulthood. There is some evidence that childhood environment explains part of the SEP differences observed in the health of young adults (Davey Smith, Blane et al. 1994). Based on the data of the British 1958 birth cohort, SEP differences in health at age 23 were not eliminated after taking account of earlier circumstances, but substantial reductions were associated

with a number of factors in childhood (Power 1991). In another British study, explanations of health inequality at age 33 spanned from early life to early adulthood (Power, Matthews et al. 1998). Overall, the evidence on the significance of different childhood factors in explaining socioeconomic health differences is scarce. Even less is known how later circumstances contribute to these effects, although it is thought that behavioural factors and living conditions play some role in the process.

Early adulthood is a significant phase of life with respect to adult health and health inequalities. For the majority of the population, this is the period of life that to a great extent determines one's educational, job and family career. Many living conditions and behavioural patterns are largely established at this stage of life. Young people experience frequent and important life transitions at least up to their thirties, and these may have profound effects on behavioural and environmental factors that are relevant to health. There has been growing research interest in the phase of transition into adulthood. New settings in adult lives provide different opportunities and norms, and formal and informal controls, as compared to the settings in adolescence. Apart from educational careers, other important life transitions include the transition from school to work, the move from family of origin to family of destination (domestic transition) and to residency away from parental home (housing transition) (Coles 1995). Health-wise, it is notable that many behavioural patterns are adopted during the first two or three decades of life, and thereafter these patterns tend to persist (McCracken, Jiles et al. 2007). As well as youth, early adulthood is often described as a period of life in which people reach a peak in terms of general health and physical fitness and when only few suffer from acute or life threatening conditions and diseases (Furlong and Cartmel 2007). However, it has been suggested that the absence of health risks is misleading as young adults suffer from health problems of their own, such as mental health problems. In addition, many lifestyle and behavioural factors (such as smoking, alcohol abuse and lack of physical activity) constitute substantial risks and have long-term consequences for health (Hurrelmann 1990). In particular, health-damaging behaviours are a major health risk among young adults, both regarding their current and later health. Socioeconomic health inequalities (Mackenbach, Bos et al. 2003; Kunst, Bos et al. 2005) also seem to emerge rapidly when heading into adulthood: they are small or non-existent in childhood and adolescence (West 1988; West and Sweeting 2004), but quite marked by early middle age (Mackenbach, Kunst et al. 1997; Valkonen, Martikainen et al. 2000; Pensola and Valkonen 2002).

Only few studies have combined information on the effects of various childhood circumstances and problems and current socioeconomic conditions as determinants of young adult health, health behaviour and health inequalities in a population-based setting. Moreover, the underlying mechanisms behind socioeconomic differences in health are yet not well understood (Adler and Ostrove 1999). The general purpose of



this study was to deepen our understanding of the development of poor health, health-damaging behaviours and health inequalities in the life-course. The aim was to find out which factors in earlier and current circumstances determine health, the indicators of the most health-damaging behaviours, and educational health differences in young adults in Finland. Following the ideas of social pathway theory, it was assumed that childhood environment affects adult health and its proximal determinants via different paths, including educational, work and family careers. The analysis covers the determinants of smoking, heavy drinking, overweight and obesity. Smoking and drinking are the most detrimental health behaviours, and in this thesis obesity and overweight are used as a proxy for the balance between nutrition and exercise, as it has been suggested that obesity is largely a consequence of over-nutrition and under-activity (Lawlor and Chaturvedi 2006).

This study increases our understanding of the development of health in the life-course and analyses the role of childhood circumstances in this process. It also adds to our knowledge about the determinants and nature of health differences in young adults. In this way it provides important background information for more successful health promotion and disease prevention aimed at reducing the risk of ill-health and health inequalities in today's young adults and tomorrow's middle-aged and elderly population. The borders between childhood, youth, adulthood and old age in the life-course are not clear and unambiguous as they are defined differently in different times and places and in different cultural contexts. In this thesis, childhood and youth cover the years before age 18, and early adulthood refers mainly to aged 18–29, but in some analyses to 18–39 years.

## **2 HEALTH AND HEALTH INEQUALITIES IN THE LIFE-COURSE**

The life-course approach to health and health inequalities emphasises that the beginnings of life, both biological and social, have important implications for the individual's potential for adult health. Biological programming may set the operational parameters for certain organs and processes, however, the primary social factors may influence the processes of biological development. They are also the beginnings of socially determined pathways to health in adult life (Wadsworth 1997). The life-course may be regarded as combining biological and social elements which interact with each other (Blane 1999). The following outlines the main characteristics of the life-course approach and the most important youth paths, both of which are adopted as the theoretical approach for this study.

### **2.1 Pathways from childhood to adult health**

Ever since the first half of the twentieth century there has been considerable epidemiological interest in the idea that early life experiences influence adult vitality and mortality risk. These ideas emerged in both the biological and psychological sciences, which emphasised the relative contribution of heredity and early environment on adult morbidity. However, the epidemics of coronary heart disease (CHD) and lung cancer in the inter-war period turned the focus of interest to the aetiology of specific chronic diseases. For several decades, the emphasis in research was on adult morbidity and lifestyle risk factors of poor health (such as smoking, drinking, poor diet and lack of physical exercise), which have also been at the centre of public health interest (Kuh and Davey Smith 1997).

In the late 1970s, however, increasing attention was given to the impact of the life-course and childhood circumstances as determinants of poor health and morbidity in adulthood (Forsdahl 1977; Forsdahl 1978). Furthermore, besides the prevailing aetiological model, researchers in the 1990s became even more interested in the life-course approach: research was showing that poor growth and development as well as adverse early environmental conditions were associated with an increased risk of adult disease. A good reason to challenge the prevailing model was provided by the extensive research carried out by David Barker and his colleagues. Basically, their argument was that different environmental factors “programme” particular body systems during critical periods of growth in utero and infancy, which may have a long-term impact on the risk of adult chronic disease (Barker, Forsen et al. 2001; Barker, Eriksson et al. 2002; Barker, Forsen et al. 2002). Since this work by Barker, there has been extensive research in epidemiology based on the life-course perspective.

There are two main ways in which different aspects of the primary social environment can affect adult health. First, childhood circumstances may affect exposures to either known or suspected causal factors during gestation, infancy, childhood, adolescence and early adulthood that are part of the long-term biological chains of risks. Second, childhood circumstances may form a part of social chains of risks that operate via educational and other experiences and lead to adult socioeconomic circumstances that affect health and the risk of disease through exposures to causal factors in later life (Kuh, Power et al. 1997). Theoretically, it is possible to distinguish between two life-course approaches: biological programming and social pathways (Power and Hertzman 1997). Interrelationships between these two approaches are, however, very complex. The pathway model that links early life and adult health takes into account the relationships between social and biological risks throughout the life-course (Power and Hertzman 1997). The origins of adult disease may lie in specific critical or sensitive periods typically in early life, or in the accumulation of detrimental exposures throughout the life-course (Kuh and Ben-Shlomo 2004).

### 2.1.1 Biological pathways: biological programming and latency model

According to biological pathways, the factors that trigger disease are either genetic or biological in nature. They affect morbidity after a latent period independently of later experiences, or in interaction with later risks. For example, David Barker discovered in the late 1980s that men who were born small had a higher incidence of heart disease decades later. He generated a number of hypotheses to explain how undernutrition during different trimesters of pregnancy programmes the individual's adult risk of disease (e.g. CHD, stroke, and diabetes mellitus). Low birth weight has been consistently shown to be associated with morbidity and risks of morbidity (Rich-Edwards, Stampfer et al. 1997; Barker, Forsen et al. 2001; Barker, Forsen et al. 2002). Biological risk factors at different stages of the life-course can have independent or interactive effects on adult disease (Kuh and Ben-Shlomo 2004).

Explanations based on biological pathways fall into two main areas. First of all, according to the latency model, a specific event or exposure in early life (before or after birth) programmes the subsequent development of disease. The latency model indicates that there are critical periods for the development of specific tissues. For example, disturbances in the growth of an infant (before or after birth, due to undernutrition, for example) have an effect on later disease only if they occur during a short but critical period. However, a person is diseased later in life regardless of exposures later in life and subsequent factors may only modify the effect. Biological programming, on the other hand, takes into account the development of disease risk

in different phases of life, not necessarily associating this risk with specific critical periods in disease initiation (Pensola 2004).

### 2.1.2 Social pathways: pathways and accumulation

Besides biological chains of risks throughout the life-course, there is another way in which socioeconomic factors and circumstances affect adult health and disease. The social pathway model can be divided into two elements: the pathway model and the accumulation model (see e.g. Pensola 2004). The pathway model emphasises that the effects of childhood economic and social hardship, for example, on subsequent adult health are partly mediated through youth paths, education and employment, and through adult circumstances (Lundberg 1993; Lundberg 1997; Pensola and Martikainen 2003; Mäkinen, Laaksonen et al. 2006). It indicates that social influences and living conditions in early life directly or indirectly determine adult health, or together with adult experiences determine adult health.

The theory of social pathways between childhood and adult health emphasises the social chains of risk that are at work throughout one's life-course (Kuh, Power et al. 1997; Mheen van de 1998; Davey Smith, McCarron et al. 2001). This theory provides the framework for this study (e.g. Kuh et al. 2004, Mheen et al. 1998b, and Davey Smith et al. 2001). The idea of social pathways is that socioeconomic factors form an integral part of social chains of risks, which starts with a socially compromised start to life, operates throughout the life-course partly via educational and other learning experiences, and leads to adult socioeconomic circumstances which affect disease risk through exposures to causal factors in later life. These causal factors include physical exposures and behavioural factors (Kuh, Power et al. 1997). According to the model, early living conditions and environments affect the pathways, such as education, which lead to adult positions. These paths mediate the effects of early circumstances on health, but at the same time they may modify them. It has also been suggested that the foundations of social inequalities in adulthood are built from these environmental and behavioural elements in early life and early adulthood (Lundberg 1993; Rahkonen, Arber et al. 1997; Davey Smith, Hart et al. 1998).

Following the ideas of this framework, it can be assumed that childhood environment affects adult health and its proximal determinants via different pathways. The positive factors in childhood environment are likely to be conducive to good health (Power, Stansfeld et al. 2002). Childhood circumstances set the trajectory into adulthood. For example, it has been suggested that parental social class is associated with educational aspirations (Koivusilta, Rimpelä et al. 1995; Power and Matthews 1997) and employment paths (Pensola 2004). The family's socioeconomic circumstances are closely related to children's educational opportunities and educational career (Wadsworth

1997), and educational level has been found to be associated with subsequent occupation, income and adult environment in general. Educational attainment and occupation are also important because of their associations with different health behaviours in childhood, adolescence and in adult life.

Risk factors for poor health appear throughout the life-course, and over time they may gradually accumulate (Ben-Shlomo and Kuh 2002; Kuh and Ben-Shlomo 2004). Accumulation may be characterised as an underlying social process driving life-course trajectories (Blane, Netuveli et al. 2007). According to the accumulation model, advantages and disadvantages may accumulate over time and have an effect on health. Childhood circumstances may then form the basis for the later accumulation of unfavourable social and economic exposures (Hertzman 1999). The accumulation hypothesis proposes that the longer the duration of exposure to disadvantaged socioeconomic position, the greater the risk of poor health. However, it has been suggested that “how” and “when” accumulation occurs has a role as well (Ljung and Hallqvist 2006). For example, as Wadsworth (1999) has conceptualised the family determinants of health from the accumulation point of view, the primary environment sets trajectories into adulthood by family environment and family function. Firstly, poor family circumstances in childhood are often associated with parental smoking, poor nutrition and low parental interest in their offspring’s education. This, in turn, may be followed by an increased risk of poor physical development in childhood as well as low educational attainment. Poor education is likely to be followed by poor socioeconomic circumstances in adulthood, poor skill attainment, unemployment as well as health damaging behaviours. Secondly, poor family function, family cohesion, poor parenting and low parental self-esteem may increase the risk of poor educational attainment, but also of poor self-control and aggressive behaviour. These, in turn, may lead to own marital breakdown, low self-esteem and poor coping strategies in adult life (Wadsworth 1999).

## **2.2 Mechanisms behind health inequalities**

Socioeconomic health inequalities based on either education, occupation or income, are well established (Mackenbach, Stirbu et al. 2008). Various models based on theories of causation, selection and their modifications have been proposed to explain these inequalities, but the reasons and mechanisms involved are still not properly understood (Adler, Boyce et al. 1994; Macintyre 1997; Bartley 1998; van de Mheen 1998). One of the first attempts to unravel these mechanisms was the Black Report in the 1980s (Townsend and Davidson 1982). This approach adopted in the report represented the traditional explanation where socioeconomic health inequalities were thought to derive from two main mechanisms: the selection mechanism and the causation mechanism. However, it was soon argued that although important, causation

and selection as such would not suffice to explain socioeconomic differences. Indeed, in the debate and discussion that followed the publication of the Black Report researchers began to consider the possibility of more complex mechanisms, including the effect of psychosocial factors as well as the development of health inequalities over the life-course.

The selection mechanism involves the impact of health in earlier life on the attainment of socioeconomic position later in life (Townsend and Davidson 1982). According to this explanation, healthy people move up in the social hierarchy, whereas unhealthy people may move down in this hierarchy: health inequalities thus occur as a result of selection in relation to health occurring during social mobility. For example, illness during childhood and adolescence may influence the attainment of adult SEP. From the point of view of direct selection, the individual's opportunities for education are crucial; poor health or illness in childhood may reduce these opportunities. Indirect selection, however, refers to a situation where poor health and low SEP both result from a third factor. For example, indirect selection based on health behaviour in adolescence may contribute to SEP differences in health (Koivusilta, Rimpelä et al. 2003).

Social causation suggests that socioeconomic status has an effect on health through unequal distribution of determinants of health across socioeconomic groups (Townsend and Davidson 1982). This means that socioeconomic status influences health through more specific determinants of health and illness, which can be called intermediary factors. Causal mechanisms are often regarded as the main explanation for socioeconomic differences. There are two main lines of explanation, material (or structural) and behavioural. However, explanations of socioeconomic differences in health referring to material and behavioural factors are not in fact separate issues since behavioural factors, for example, are partly embedded in a number of material and structural living conditions. People have access to different material conditions depending on their socioeconomic position. This refers to the effects of poorer material conditions on health (for example poor housing or work-related conditions and hazards) or relative deprivation (where people assess their own SEP in relation to others, irrespective of absolute affluence). It is likely that the origin of inequalities in health lies partly in the fact that people in lower socioeconomic groups live and work in circumstances that may have a detrimental effect on health. The behavioural explanation indicates that those in lower socioeconomic positions have poorer health due to health-damaging behaviours (smoking, drinking, physical inactivity, infrequent use of health care, etc.), which are more common in lower than in higher socioeconomic groups (Townsend and Davidson 1982).

In causation explanations, not only material and behavioural factors but also psychosocial and stress-related factors have received much attention as possible

explanations for health inequalities. It has been suggested that the distribution of psychological stress is an important determinant of health inequalities in today's affluent societies (Macintyre 1997; Elstad 1998). It may be expected that people in lower socioeconomic strata are more exposed to stressful circumstances, or are less capable to cope with these stressors. As a result, they may be more prone to the negative effects on health. The psychosocial perspective supports the idea that psychosocial pathways are associated with relative disadvantage, which adds to the direct effects of absolute material living conditions (Wilkinson 1996; Marmot and Bobak 2000; Marmot, Shipley et al. 2001). This perspective focuses on the psychosocial impact of stress-related inequality structures, induced psychologically as well as materially. The two different pathways from stress to health are first, the direct effect of stress on disease development and second, an indirect route where stress leads to health damaging behaviours. However, it has also been suggested that a psychosocial interpretation of health inequalities, in terms of perceptions of relative disadvantage and the psychological consequences of inequality, may give rise to several conceptual and empirical problems (Lynch, Smith et al. 2000).

Over the past decades it has been increasingly emphasised that it is not only current SEP but also the primary social background that has an impact on health inequalities. Regarding the effect of the life-course, socioeconomic health differences in adult life could partly derive from processes in earlier life. The key question here is whether those with the lower SEP are less healthy because they have grown up in a less advantaged environment, or whether the effect of childhood is independent of adult socioeconomic position. Both causation and selection mechanisms have roles in this process. The life-course perspective on health inequalities involves the accumulation of adverse socioeconomic circumstances and selection, which may cause a downward spiral (van de Mheen, Stronks et al. 1998). The influence of childhood social circumstances on adult health differences may be due to social programming, where the effects on adult health are mediated through social conditions, education, entry into work as well as health behaviours and lifestyle. Another process, a selection process, involves the effect of childhood health on health inequalities in adulthood; in this case persons with lower SEP in adulthood may be less healthy because of their poorer health in childhood (van de Mheen, Stronks et al. 1998). However, it is assumed that the contribution of childhood circumstances to the gradient in health occurs through the combination of latent effects, pathway effects and cumulative disadvantage (Hertzman 1999).



## 2.3 Youth paths and health

Adverse childhood circumstances may influence opportunities in education, job opportunities and life changes in general, resulting unhealthy life careers (Lundberg 1993). Important transitions take place when entering adulthood. For example, educational careers, the transition from school to work, the move from family of origin to family of destination (domestic transition) and to residency away from parental home (housing transition) can be regarded as important transitions which also have health implications (Coles 1995). Youth paths may mediate the effect of circumstances in childhood on adult health. An adult risk factor or exposure may mediate the association between childhood exposure and health outcome when it chronologically follows the exposure and is conceptualised as lying, at least partly, on the causal pathway (Kuh, Ben-Shlomo et al. 2003). From the point of view of this thesis, three potential pathways should be mentioned: educational path, employment path and family formation path. It has been suggested that poorer conditions in parental home are associated with less favourable youth paths, which may further lead to a lower social class and early family formation in adulthood and to poorer health (Pensola 2004).

Perhaps the most frequently hypothesised pathway between primary social circumstances and adult health is education, which is often the first dimension of SEP that is established in the life-course. First of all, adult education can be considered a potential pathway because it is heavily influenced by primary SEP (Pöntinen 1983; Power and Matthews 1997; Koivusilta 2000; Pensola 2004), and it is itself predictive of many subsequent health outcomes, as described in detail in Chapter 3. It has been suggested that parental social class is associated with educational aspirations even in adolescence (Koivusilta, Rimpelä et al. 1995; Power and Matthews 1997). Socioeconomic family circumstances are also closely related to the child's educational opportunities and educational career (Wadsworth 1997). For example, emotional disruption in the family can reduce the child's likelihood of high educational attainment. Parental divorce and separation have also been shown to be associated with reduced educational attainment (Ely, Richards et al. 1999). Part of the reason why educational attainment is so important is that it has been found to be associated with subsequent occupation, income and adult environment in general. Educational attainment and occupation are also important because of their associations with different health behaviours in childhood and adolescence and in adult life. It has been suggested that educational paths are an essential part of social programming from parental home to adult social class, which mediates the effect of parental home for example on mortality in middle adulthood (Pensola 2004).

However, educational career is not the only pathway linking primary SEP and childhood circumstances to adult health. Childhood circumstances and primary SEP



may also affect the individual's employment path. According to a Finnish study based on register data, unemployed men and women were more likely to have an adverse social background than others, i.e., a single-parent family and unskilled manual social class background increased the risk of unemployment, for example (Pensola 2004). Similar findings have been presented from the British 1946 and 1958 birth cohorts (Wadsworth, Maclean et al. 1990; Power and Matthews 1997). Unemployment has associations with health outcomes as well as health damaging behaviours, as described later, and therefore potentially mediates the effects of childhood circumstances on adult health as well.

It has been found that parental social class and social environment are also associated with family formation. In particular, it seems that early marriage and having children at young ages is common in people with lower primary SEP and from single-parent homes (Kuh and Maclean 1990; Pensola 2004). Childhood family structure and parental adversity may also affect living arrangements in adulthood, as indicated by a recent Finnish study in the adult population. In addition, several health outcomes depend on family structure and living arrangements in adulthood (Joutsenniemi, Martelin et al. 2006).

### **3 CHILDHOOD CIRCUMSTANCES AND LATER HEALTH: A REVIEW OF THE LITERATURE**

This chapter reviews the recent international literature and relevant research findings on childhood determinants of health, health behaviour and health inequalities. In addition to the literature based on datasets and cohorts from Finland (e.g. The Northern Finland 1966 Birth Cohort, LASERI, HeSSup, HHS, TAM, Health 2000) and several other Western countries, two key sources should be given separate mention. First, the earlier literature on the contribution of childhood and current circumstances to young adult health and health inequalities, based on longitudinal datasets often refers to the reliable and valuable analyses of British birth cohorts (e.g. 1946 and 1958). However, it is noteworthy that the determinants of health may be very different due to socio-cultural differences between Finland and Britain and between the samples studied. People born in 1946 or 1958 in Britain will probably have lived their childhood in a very different sociocultural environment than the population of young adults in Finland born in the 1970s and in the early 1980s. However, bearing these differences in mind, the studies provide a valuable background for the analyses of this thesis.

Although not fully comparable with Finnish data due to differences in age ranges, another important dataset is the Adverse Childhood Experiences (ACE) Study, a long-term, in-depth analysis of over 17,000 adult Americans which matches their current health status against adverse childhood experiences. The reason it is important to this thesis is because it is a retrospective cohort survey and because there is an extensive literature based on this dataset. The ACE Study is interested to analyse the relationships between multiple categories of childhood trauma (ACEs) and health and behavioural outcomes later in life. The ACE Study was conducted in 1995–1997, which means that some consideration must be given to the issue of socio-cultural time and the nature of experienced childhood.

#### **3.1 Childhood and current circumstances as determinants of adult health**

Social and economic circumstances in the parental home, such as parental SEP, material deprivation and parental unemployment, family type and social adversities, may influence health in adulthood directly or indirectly by influencing youth paths, which in turn affect health. In addition, both childhood and current circumstances can together affect subsequent health. This section reviews previous research findings on the association between childhood circumstances and health, both physical and mental.

### 3.1.1 Self-rated health, morbidity and mortality

The impacts of childhood circumstances on adult health have been observed in several studies for several indicators of health, such as self-rated health and chronic diseases (Kaplan and Salonen 1990; Lundberg 1993; Lundberg 1997; Rahkonen, Lahelma et al. 1997; Bosma, van de Mheen et al. 1999; Dube, Felitti et al. 2003; Dong, Giles et al. 2004), disability pension (Gravseth, Bjerkedal et al. 2007; Harkonmäki, Korkeila et al. 2007) as well as mortality (Lynch, Kaplan et al. 1994; Davey Smith, Hart et al. 1998; Davey Smith, McCarron et al. 2001; Claussen, Davey Smith et al. 2003; Pensola 2004). The discussion below reviews the previous literature on predictors of self-rated health, which has been claimed to reflect physical health (Ratner, Johnson et al. 1998), but also other dimensions of well-being. It also describes some corresponding findings on morbidity and mortality. In general, the impacts of earlier life on adult physical health comprise a wide range of factors, including biological and environmental effects, as well as both earlier and later life circumstances.

#### Childhood predictors of self-rated health, morbidity and mortality

Primary SEP has been found to be associated with later health. In general, the lower the SEP in childhood, the poorer the health later in life. For example, an earlier study of young adults in Finland and Britain found a relatively weak but consistent effect of low primary SEP on both self-rated health and long-standing illness (Rahkonen, Arber et al. 1995). However, other studies on young adults in Finland have indicated that lower parental SEP has no impact on physical health (health status and chronic illness) (Huurre, Aro et al. 2003). Power and colleagues found in the British birth cohort that SEP from birth to 33 years of age had a cumulative effect on poor self-rated health in early adulthood (Power, Manor et al. 1999). In addition to poor SRH, several studies have recognised the influence of low SEP throughout the life-course on risk of disease in adulthood (Galobardes, Lynch et al. 2004; Melchior, Moffitt et al. 2007).

Various adverse childhood circumstances have been found to be associated with several adult diseases, particularly with cardiovascular disease and its risk factors (Forsdahl 1977; Forsdahl 1978; Barker et al. 1986; Hasle 1990; Kaplan et al. 1990; Elford et al. 1991; Wannamethee et al. 1996; Barker et al. 2002b; Poulton et al. 2002; Claussen et al. 2003; Dong et al. 2004; Galobardes et al. 2004; Sumanen et al. 2005; Galobardes et al. 2006; Kittleson et al. 2006; Sumanen et al. 2007), although for some health outcomes (such as allergies) no associations has been found (Bergmann, Edenharter et al. 2000). For example, it has been found that adverse socioeconomic position across the life-course cumulatively increases CHD risk, and this effect is not fully explained by adult risk factors (Lawlor, Ebrahim et al. 2005). Besides CHD, adverse childhood experiences have been found to be associated with chronic liver

disease (Dong, Dube et al. 2003), type 2 diabetes mellitus (Agardh, Ahlbom et al. 2007), midlife functional status (Guralnik, Butterworth et al. 2006) and limiting long-standing illness (Power, Li et al. 2000).

Childhood family structure has been found to be important in relation to later health, as those from single-parent backgrounds seem to have worse health in adulthood. Lack of household resources potentially plays a role in this increased risk. However, even when a wide range of demographic and socioeconomic circumstances are included in multivariate models, children of single parents still have increased risks of severe morbidity (Ringsbäck-Weitoft, Hjern et al. 2003). Regarding family structure otherwise, the number of siblings (as a possible indicator of living conditions in childhood) associates with health in adulthood, for example with gastric cancer risk (La Vecchia, Ferraroni et al. 1995), which probably indicates infections acquired in childhood.

In addition, reports have been published on the effect of specific childhood adversities on poor adult SRH and illness. In a Swedish study by Lundberg from the early 1990s, it was found that conflicts in the family during upbringing were strongly related to illness later in life. Living in a broken family and, to some extent, economic hardship during childhood were clearly associated with illness later in life. This finding did not change even when controlling for age, gender and paternal SEP (Lundberg 1993). In a Finnish study, however, financial problems were stronger and more independent determinants of adult SRH than were social problems. Living conditions during upbringing, particularly financial problems and status of origin, were significant predictors (Rahkonen, Lahelma et al. 1997). Parental long-term unemployment (especially that of fathers) has been found to be negatively associated with at least adolescents' SRH. Father's long-term unemployment was a significant predictor of moderate SRH and low long-term well-being in men and women, and mother's long-term unemployment was negatively associated with SRH of women and longstanding illness in men (Sleskova, Salonna et al. 2006). However, it has been concluded that the link between parental employment status and the health of their children may vary between countries (Sleskova, Tuinstra et al. 2006).

Among biological childhood predictors of poor health in adulthood, low birth weight has consistently shown to be associated with CHD and its biological risk factors. Barker and colleagues have shown that the combination of small size at birth and during infancy followed by accelerated weight gain from age 3 to 11 years predicts large differences in the cumulative incidence of CHD, type 2 diabetes mellitus and hypertension later in life (Barker, Eriksson et al. 2002). Hypertension originates in slow foetal growth followed by rapid growth in childhood. These biological factors have been found to interact with environmental factors. For example, the path of growth has a greater effect on the risk of disease in children who live in poor

social conditions. However, circumstances in adulthood do not seem to be important (Barker, Forsen et al. 2002). It has also been found that socioeconomic environment has an impact on small body size: men who grow slowly in utero remain biologically different to other men and are more vulnerable to the effects of low SEP and low income on CHD (Barker, Forsen et al. 2001). Barker has emphasised the long-term effects of infant deprivation on adult health, but more critical views have also been published (Vågerö and Leon 1994). There is consistent evidence on the relationship between small body size at birth and during infancy and later cardiovascular disease and its risk factors (Barker, Winter et al. 1989; Rich-Edwards, Stampfer et al. 1997) and diabetes mellitus (Rich-Edwards, Colditz et al. 1999). Also, those with lower birth weight have been found to have poorer educational and cognitive outcomes in early adulthood (Lefebvre, Mazurier et al. 2005).

To divert briefly from subsequent health and morbidity to mortality, the association between adverse social circumstances and higher risk of mortality in adulthood has been demonstrated, again, primarily for cardiovascular causes of death (Forsdahl 1978; Barker and Osmond 1986; Lynch, Kaplan et al. 1994; Vågerö and Leon 1994; Davey Smith, Hart et al. 1998; Pensola and Valkonen 2002; Pensola and Martikainen 2003; Pensola 2004; Power, Hypponen et al. 2005; Strand and Kunst 2006; Strand and Kunst 2007). A recent systematic review (Galobardes, Lynch et al. 2008) on the associations between childhood socioeconomic circumstances and cause-specific mortality (covering studies published since 2003) confirmed that mortality risk was higher in those who experienced poorer socioeconomic circumstances during childhood. According to this review, education was an important mediator between early life socioeconomic position and adult mortality. However, the relative importance of primary and current SEP is not clear and it seems to depend on the cause of death as well (Davey Smith, Hart et al. 1998; Beebe-Dimmer, Lynch et al. 2004; Naess, Strand et al. 2007). For example in Norway, cardiovascular disease mortality was found to be more strongly associated with childhood than with adulthood social circumstances, while the opposite was found for psychiatric and accidental/violent mortality (Claussen, Davey Smith et al. 2003). Some studies, however, indicate that socioeconomic conditions in childhood are not important determinants of mortality in adulthood in the first place (Lynch, Kaplan et al. 1994). In the mortality of young adults, low primary SEP has been found to be associated with an increased risk for most causes of death (Strand and Kunst 2007).

#### Adult risk factors and potential pathways to poor self-rated health, somatic morbidity and mortality

Children from socioeconomically disadvantaged families may be more likely to be born with physical health problems due to poorer nutrition, maternal smoking and

other similar factors. Secondly, it is likely that unfavourable childhood circumstances are associated with poorer ability to provide proper nutrition and adequate access to health care for children, for example. Poor parental practices as well as poor health and detrimental behaviour may contribute to poorer health of the child. Long-term exposure to stressful childhood experiences may also exert direct effects on biological functioning. Finally, family adversities impose structural constraints on choices regarding health-related behaviours that can result in an unhealthy lifestyle, for example (Wickrama, Conger et al. 1999).

Adult determinants, such as educational tracks and other youth paths, may mediate the effect of childhood circumstances on adult health. However, the evidence on the relative importance of childhood and current circumstances on adult health is inconsistent. Some studies have indicated that adverse SEP in childhood is associated with poorer health independently of adult SEP and across diverse measures of disease risk and physical functioning (Power, Atherton et al. 2007). However, The Whitehall II Study found that adult SEP was a more important predictor of mortality attributable to coronary disease and chronic bronchitis than measures of social status earlier in life. According to that study, social circumstances early in life may influence employment and SEP and thus exposures in adult life (Marmot, Shipley et al. 2001). For example, there is a lot of evidence that the parental SEP is associated with youth paths and adult SEP (Power and Hertzman 1997; van de Mheen, Stronks et al. 1997; Pensola and Martikainen 2004), which in turn affect health. In a recent Finnish study (HHS), childhood circumstances were not directly associated with physical functioning in the adult population but had some effect via the respondent's own SEP (Laaksonen, Silventoinen et al. 2007).

A study based on the 1958 British birth cohort presented an integrated model of the determinants of adult SRH, combining life-course factors and contemporary circumstances, and explored the latent, pathway and cumulative effects. According to the findings, the effects of childhood circumstances were not removed by the inclusion of contemporary factors, and conversely, contemporary factors contributed to the prediction of SRH over and above life-course factors. The authors concluded that both life-course and contemporary circumstances should be considered together in the explanations (Hertzman, Power et al. 2001). In addition, a follow-up study from New Zealand investigated which factors contribute to an excess risk of poor health at age 32 in those who experienced socioeconomic disadvantage in childhood. These results showed that low childhood SEP was associated with an increased risk of poor physical health (cardiovascular risk factor status) in adulthood, and it was suggested that the processes mediating the link between low primary SEP and poor adult health are multifactorial (Melchior, Moffitt et al. 2007). Some other studies have also indicated that primary SEP and accumulated disadvantage constitute a distinct socioeconomic

influence on poor adult poor health (Turrell, Lynch et al. 2007). According to a Norwegian study on mortality in young adults, the effect of childhood circumstances seems to depend on the cause of death: primary SEP had a direct association with early adult cardiovascular mortality in men, whereas for other causes of death primary SEP showed only an indirect association, mostly through individuals persons' own educational level (Strand and Kunst 2007). In Finland, the effect of parental class on the mortality of young men has also been found to be indirect and mainly mediated through its influence on education and SEP (Pensola and Valkonen 2002).

The effect of childhood circumstances on adult health, independent of adult SEP, may also operate partly through unhealthy behaviour (van de Mheen, Stronks et al. 1998), as it has been found to mediate the association between parental SEP and adult disease risk (Pensola and Valkonen 2000). The causal mechanisms between adverse childhood experiences and adult illness include factors related to lifestyle, such as smoking, diet and physical activity (Blane, Hart et al. 1996; Lynch, Kaplan et al. 1997; van de Mheen, Stronks et al. 1998). Childhood socioeconomic circumstances have an independent effect on adult health-related behaviour; in general, the risk of unhealthy behaviours is higher in lower childhood socioeconomic groups. However, not all studies have found the effect. A study based on the 1946 British birth cohort set out to establish whether adulthood behavioural risk factors explained the association between childhood SEP and midlife physical function. According to the results, early adulthood behavioural risk factors and middle-age SEP and disease status only modestly attenuated the relationship between paternal SEP and low physical functioning (Guralnik, Butterworth et al. 2006).

Some work has also been done to explore the role of psychosocial factors as potential mediators between childhood adverse circumstances and poor health. In a Dutch study, a higher prevalence of negative personality profiles and adverse coping styles in subjects who grew up in lower social classes explained part of the association between low SEP in childhood and adult poor self-rated health (Bosma, van de Mheen et al. 1999). In a Swedish study, on the other hand, sense of coherence did not mediate the effect of childhood circumstances on adult health. Rather, poor childhood conditions and low sense of coherence in adulthood appear to be complementary and additive risk factors for illness in adulthood (Lundberg 1997).

Some differences have been found in self-reported health by degree of urbanisation at place of residence. Recent results from the Northern Finland 1966 birth cohort show that poor self-reported health and general dissatisfaction with life is more common in rural areas. However, this association was seen primarily for the mediating effect of unemployment, poorer education, lack of social support, passive coping strategies and greater pessimism in people living in rural areas (Ek, Koironen et al. 2008).



### 3.1.2 Mental health

Mental health research has been increasingly interested in the childhood processes that jeopardise successful transition to adulthood, which is a critical period in the course of psychopathology and mental health. Depression during the transition from late adolescence to early adulthood is a major mental health concern (Berry 2004; Schulenberg, Sameroff et al. 2004). It is one of the most disabling diseases, and causes a significant burden both to the individual and to society. Youth has always been a period of transitions that involves psychological adjustment while establishing adult identities. However, levels of depression and stress-related problems have appeared to be increasing among young people over the past few decades, at least in some countries (Smith and Rutter 1995). It has been suggested that the changes sweeping society and experiences in youth may have resulted in increased levels of stress, which are subsequently manifested in psychological ill-health (West 1996; West and Sweeting 2003; Furlong and Cartmel 2007).

It seems that exposure to adverse childhood experiences is associated with an increased risk of psychological symptoms and disorders up to decades after their occurrence (Chapman, Whitfield et al. 2004), and that these experiences interact with current adult risk factors of mental health. Previous studies have shown that childhood circumstances and adversities are associated with a variety of indicators of mental health in adolescence and adulthood, such as depressive and anxiety disorders (Felitti, Anda et al. 1998; Veijola, Puukka et al. 1998; Sadowski, Ugarte et al. 1999; Infrasca 2003; Levitan, Rector et al. 2003; Korkeila, Korkeila et al. 2005; Turner, Finkelhor et al. 2006; Brown, Craig et al. 2007; Schilling, Aseltine et al. 2007), psychotic experiences (Janssen, Krabbendam et al. 2004), antisocial behaviour (Schilling, Aseltine et al. 2007), personality disorders (Rutter and Maughan 1997; Johnson, Cohen et al. 1999; Johnson, Smailes et al. 2000; Rosenman and Rodgers 2006), decreased optimism (Korkeila, Kivelä et al. 2004), decreased cognitive, social, mental and psychosocial functioning (Lynch, Kaplan et al. 1997; Kaplan, Turrell et al. 2001; Harper, Lynch et al. 2002; Pulkkinen 2003; Mäkinen, Laaksonen et al. 2006), and suicide attempts (Dube, Anda et al. 2001).

#### Childhood predictors of mental health

Primary SEP has been found to be associated with mental health indicators later in life. A Finnish follow-up study (TAM) concluded that low parental SEP impacts early adult well-being as women from manual class backgrounds had lower self-esteem and more distress symptoms from adolescence to adulthood than those from non-manual backgrounds. Men from manual class families were found to have lower self-esteem in adolescence and early adulthood (Huurre, Aro et al. 2003). However, there are also



opposite findings for some indicators, for instance regarding suicide attempts (Strand and Kunst 2006).

Numerous studies have revealed differences in mental health by the structure of one's family of origin. In particular, there is strong evidence on the adverse effects of living in a single-parent family. Based on a large sample of young adults, a US study found higher levels of depressive symptoms in those from stepfamilies, single-parent families and single parent families with other relatives present compared to mother-father families (Barrett and Turner 2005). In addition, an analysis of 971 young adult participants with available data on exposure to single parenthood from birth to age 16 revealed significant associations between that exposure and anxiety disorders (Fergusson, Boden et al. 2007). Individuals from a single-parent family background have also been found to have an elevated risk of hospital-treated non-psychotic disorder. In this study it was concluded that a combination of the single-parent family and psychosocial and/or genetic risk probably contributes to the development of these disorders (Mäkikyrö, Sauvola et al. 1998).

The effects of specific adverse childhood experiences have been studied in relation to mental health. According to the ACE Study, childhood adversities contribute to the burden of adult mental illness (Anda, Brown et al. 2007). Parental depressive disorders are known to raise the risk of mental disorders in offspring (Mäkikyrö, Sauvola et al. 1998; Marmorstein, Malone et al. 2004; Rosenman and Rodgers 2006), and alcohol-related problems also constitute risks for depression and depressive disorders, although their effect may vary by gender. In a Finnish study on the adult population, paternal mental health problems showed a particularly strong association with depressive disorders in men and maternal mental health problems with depressive disorders in women. Maternal alcohol problems, however, were associated with alcohol use disorders in both genders (Pirkola, Isometsä et al. 2005). There is some indication that depression in adult offspring of alcoholic parents is largely due to the greater likelihood of adverse childhood experiences in a home with alcohol-abusing parents (Anda, Whitfield et al. 2002). Childhood sexual and physical abuse is associated with poor mental state later in life as well (Edwards, Holden et al. 2003). In general, adverse parenting in childhood, particularly a maternal parenting style typified by low care, is a significant risk factor for adult depression (Oakley-Browne, Joyce et al. 1995). Furthermore, parental maltreatment is a critical determinant of later chronic depressive episodes in adult women (Brown, Craig et al. 2007).

One well-researched childhood adversity in relation to later poor mental health is parental divorce, which seems to be an important risk factor (Wadsworth, Maclean et al. 1990; Rodgers 1994; Rodgers, Power et al. 1997; Huurre, Junkkari et al. 2006). A recent Finnish study based on prospective follow-up data (TAM) examined how 32-year-old young adults who had experienced parental divorce before age 16

differed in psychosocial well-being from those who had grown up in non-divorced two-parent families. The results showed that women from divorced compared to non-divorced families reported more psychological symptoms. However, these differences were not found in men. The study confirmed that parental divorce is an indicator of stress in childhood, the effect of which persists into adulthood (Huurre, Junkkari et al. 2006). Although the effect of parental divorce may depend on the child's age at which parental divorce is experienced, at least no moderating effects were found for age at separation in the association found between childhood parental divorce and adult psychological distress in a British national birth cohort at ages 23 and 33 (Rodgers, Power et al. 1997). A similar non-age-dependent finding has been reported regarding the association between parental divorce and subsequent well-being in two British birth cohorts (Sigle-Rushton, Hobcraft et al. 2005).

Childhood adversities rarely occur in isolation and they are closely interrelated (Felitti, Anda et al. 1998; Anda, Croft et al. 1999). Some studies have documented strong associations between cumulative childhood adversity (CCA) and several indicators of mental health, such as adult major depression and depressive symptoms (Turner and Lloyd 1995; Hammen, Henry et al. 2000; Anda, Whitfield et al. 2002; Turner and Butler 2003; Chapman, Whitfield et al. 2004), adolescent depressive symptoms (Turner, Finkelhor et al. 2006) and alcohol and substance use (Dube, Anda et al. 2002). The results indicate that the accumulation of childhood adversities is associated with poorer mental health in adulthood: the risk of both life-time and recent depressive disorders increases progressively with the number of reported childhood adversities (Anda, Whitfield et al. 2002). The ACE Study revealed that an increasing number of adverse childhood adversities increased the risk of poor mental health and life-time suicide attempt and had a consistent, strong and graded relationship with them (Dube, Felitti et al. 2003). In a recent Finnish study (based on the Health 2000 Survey), a moderate dose-response relationship was observed between the total number of adversities and current mental disorders in the adult population (Pirkola, Isometsä et al. 2005). Adversities may begin to have an impact on mental health even in childhood: for example, the number of life events with a negative impact has been found to correlate negatively with children's perceived self-competence (Beardsall and Dunn 1992).

School is an important life context in childhood and adolescence, and school experiences may play a strong role in the development of mental disorders. First of all, being bullied at school has been found to predict mental disorders in both adult men and women in Finland (Pirkola, Isometsä et al. 2005). The effects of childhood circumstances on mental health may be gender-specific. For example, researchers have found marked gender differences in the associations between reported childhood experiences and environmental circumstances and adulthood mental disorders. In

women, a greater number of adversities are associated with mental disorders and their statistical significance has been found to be greater than in men (Pirkola, Isometsä et al. 2005).

#### Adult risk factors and potential pathways to poor mental health in adulthood

The impact of childhood adversities on early adult mental health is probably composed of a wide range of factors from direct causal associations to complex, interacting environmental effects (Pirkola, Isometsä et al. 2005). The pathway from early life adversity to adult mental health may be direct or mediated through adult circumstances. In addition, adult risk factors can act as vulnerability factors, which means that the consequences of adverse childhood background may be worse if combined with adult negative life events (Kendler, Karkowski et al. 1999). A Finnish study based on a large sample of working-aged men and women (HeSSup) concluded that the associations between childhood adversity and depressiveness were partly mediated by adult risk factors, supporting a pathway from childhood adversities to depressiveness through adult risk factors. In another recent Finnish study (HHS), childhood circumstances were not directly associated with mental functioning in the adult population, but had some effect through socioeconomic position. In that study, mental functioning was poorer among those in higher positions (Laaksonen, Silventoinen et al. 2007). One of the possible mediating factors between childhood circumstances and later mental health is education. However, a Finnish follow-up study (TAM) concluded that parental SEP has effects on early adult well-being other than those mediated by current SEP (Huurre, Aro et al. 2003). The combination of childhood and later experiences has also been found to have an effect on mental health: respondents with childhood adversities in combination with recent death/illness events were found to have an increased vulnerability for depressiveness (Korkeila, Kivelä et al. 2004).

Unemployment seems to be an important adult risk factor for mental disorders. It is possible to think that adverse social circumstances lead not only to low education but also to unemployment, which in turn is reflected in mental health. In Germany, young adults (mean age 29 years) with more experiences of unemployment reported higher global distress, more anxiety and depression (Berth, Forster et al. 2003). According to a recent Finnish study, long-term unemployment was associated with an increased risk of major depressive episodes. Moreover, frequent alcohol intoxication in long-term unemployed individuals increases the risk of depression (Hämäläinen, Poikolainen et al. 2005). A study exploring the relationship between unemployment and health problems in men in France indicated that unemployed men were more likely to have a significantly higher prevalence of depression and consumption of psycho-active drugs than the working population (Khlat, Sermet et al. 2004). In a New Zealand

cohort of young adults, exposure to unemployment was associated with mental health indicators (depression, anxiety) when the confounders were not taken into account (Fergusson, Horwood et al. 2001). The association between long-term unemployment and psychological health has been found to be even stronger in young people than in adults (Reine, Novo et al. 2004).

Adult family structure and living arrangements may also be of importance in relation to mental health. A recent Finnish study on a representative sample of adults (Health 2000) indicated that adult living arrangements were strongly associated with mental health, particularly in men. Compared with married persons, those living alone and those living with other(s) than a partner were approximately twice as likely to experience anxiety or depressive disorders. Cohabitors did not differ from married persons. In men, psychological distress was similarly associated with living arrangements (Joutsenniemi, Martelin et al. 2006). Single parenthood has been found to be associated with elevated psychological distress in women, especially in younger age-groups. In men, no such association was found, although there was some indication of elevated distress in younger fathers (Avison and Davies 2005). A Finnish study investigating the correlates of depression in a general population sample of adolescents (15–19 years) and young adults (20–24 years) found that in young adults, not being married and not cohabiting were related to major depressive episodes (Haarasilta, Marttunen et al. 2004). However, support and other qualitative aspects of family structure are also associated with mental health in young married people (Horwitz, McLaughlin et al. 1998).

The effect of childhood social circumstances may also be mediated through psychological and psychosocial factors. In an Israeli study, emotional abuse in childhood and perceptions of controlling and non-caring parents were found to have an indirect effect on psychopathology. This was mediated by immature defences and low self-esteem. It was concluded that psychopathology in students (aged 20–45) who suffered emotional abuse in childhood was produced by the effect of abuse on personality, taking the form of immature defence organisation and damaged self-representation (Finzi-Dottan and Karu 2006). In another study the effects of childhood circumstances on suicidal behaviour in young people aged 15–21 were largely mediated by mental health problems and exposure to stressful life events during adolescence and early adulthood. The risk of developing suicidal behaviour, however, depends on accumulative exposure to a series of social, family, personality and mental health factors (Fergusson, Woodward et al. 2000).

## **3.2 Development of health-damaging behaviours and obesity in the life-course**

Health behaviours are important determinants of health and health inequalities (Townsend and Davidson 1982; Stronks, van Trirum et al. 1996). Health-damaging behaviours such as eating poorly, being physically inactive, using large amounts of alcohol and smoking contribute substantially to the leading causes of morbidity and mortality and are often established during adolescence and early adulthood (McCracken, Jiles et al. 2007). An expanding body of research suggests that childhood circumstances can lead, beside to and potentially before the negative health outcomes, to health-damaging behavioural habits. The existence of socioeconomic differences in health-related behaviour has made clear that behaviours occur in social contexts and are also a response to the socioeconomic circumstances (Jarvis and Wardle 1999).

The reasons why individuals adopt certain behaviours are said to include the influences of earlier life experiences, the social and economic environment and the characteristics of the individual (Droomers 2002). Previous findings show that many adult health-damaging behaviours are related to low SEP and poor childhood conditions (Lynch, Kaplan et al. 1997). The ACE Study has shown a strong graded relationship between the number of childhood adversities and health behaviours and problems related to behavioural factors, such as smoking (Anda, Croft et al. 1999), adolescent pregnancy (Hillis, Anda et al. 2004), sexual risk behaviours and sexually transmitted diseases (Hillis, Anda et al. 2000; Hillis, Anda et al. 2001), male involvement in teen pregnancies (Anda, Felitti et al. 2001), adult alcohol problems (Anda, Whitfield et al. 2002; Dube, Anda et al. 2002), illicit drug use (Dube, Felitti et al. 2003) and many leading causes of death (Felitti, Anda et al. 1998) in the United States. The following sections describe earlier research findings on the life-course determinants of the most health-damaging behaviours (smoking, heavy drinking and obesity as a proxy of the balance between nutrition and physical activity).

### **3.2.1 Smoking**

Smoking is the most common preventable cause of premature morbidity and mortality (Peto, Lopez et al. 1994) and an important pathway for the emergence of poor adult health and health inequalities (Power and Hertzman 1997; Power and Matthews 1997; Schrijvers, Stronks et al. 1999). Smoking is one of the most constant health behaviours from adolescence to adulthood (Paavola, Vartiainen et al. 2004), and the period from mid-adolescence to early adulthood is also important for uptake of regular smoking (West, Sweeting et al. 1999). The majority of smokers start smoking as teenagers (West, Sweeting et al. 1999). The dependency then deepens during early adulthood

and continues until the habit is possibly quit in middle age (Peto, Darby et al. 2000). A significant number of young people use tobacco during their teenage years (Naidoo, Warm et al. 2004); this applies to Finland as well (Rimpelä, Lintonen et al. 2002). In general, tobacco use has been found to be relatively high in the youngest age groups, peaking in young adults and declining at older ages (Anthony and Echeagaray-Wagner 2000). A recent US study suggests that socioeconomic inequalities in smoking also emerge in early adulthood (Yang, Lynch et al. 2008).

The prevalence of daily smoking is determined by the incidence of smoking initiation, maintenance and the quit rate. The development of tobacco dependency from situational social bonding to a physiological and psychological dependency syndrome is a long process (Benowitz 1998) that is affected by both environmental and genetic factors (Li 2003; White, Hopper et al. 2003), although it has been suggested that symptoms of tobacco dependence develop rapidly (DiFranza, Savageau et al. 2002). Initiation, maintenance and cessation all have strong social gradients with both childhood and adulthood socioeconomic circumstances (van de Mheen, Stronks et al. 1998; Broms, Silventoinen et al. 2004; Laaksonen, Rahkonen et al. 2005; Power, Graham et al. 2005). The family unit is the primary source of transmission of basic social, cultural, genetic and biological factors that may underlie smoking. Familial and early life influences have been identified as key determinants of smoking initiation and adolescent smoking behaviour. However, these influences on the risk of persistent smoking may differ from those found to influence smoking initiation (Madden, Heath et al. 1999).

Smoking plays a central role in the associations between health behaviours and has been found to be predictive of most other health-damaging behaviours (Prättälä, Karisto et al. 1994; Laaksonen, Luoto et al. 2002). Unhealthy behaviours accumulate to a much lesser extent in non-smokers than in smokers, which implies that smokers are probably consistent in their unhealthy behaviour. For example, smoking has been found to be associated with both unhealthy alcohol use and physical inactivity (Paavola, Vartiainen et al. 2001; Paavola, Vartiainen et al. 2004). A Finnish-Swiss comparison showed that a consistent cross-cultural pattern of health-related behaviours can be detected even in young people aged 16 and 18 years (Karvonen, Abel et al. 2000).

### Childhood predictors of smoking

Low parental SEP has been found to be associated with smoking in adolescence and adulthood (Green, Macintyre et al. 1991; Scarinci, Robinson et al. 2002; Huurre, Aro et al. 2003; Jefferis, Graham et al. 2003; Jefferis, Power et al. 2004; Naidoo, Warm et al. 2004; Droomers, Schrijvers et al. 2005; Fagan, Brook et al. 2005). In young Finnish adults (TAM), it has been found that smoking is more prevalent in those coming from

a manual class of origin than in those from other social classes (Huurre, Aro et al. 2003). A US study also reported an increased risk of smoking initiation, progression to regular smoking and a reduced likelihood of smoking cessation in adults from lower socioeconomic backgrounds (Gilman, Abrams et al. 2003). However, there are also studies that have reported no or only inconsistent associations between primary SEP and adult smoking (Blane, Hart et al. 1996; Tuinstra, Groothoff et al. 1998; Paavola, Vartiainen et al. 2004).

Several specific childhood adversities have been found to be risk factors for smoking in adulthood. The ACE Study revealed that adverse childhood experiences were associated with smoking in adulthood as compared to those reporting no adverse childhood experiences (emotional, physical and sexual abuse; a battered mother; parental separation or divorce; and growing up with a substance-abusing, mentally ill, or incarcerated household member), persons reporting five categories or more experiences, had a substantially higher risk of early smoking initiation, ever smoking, current smoking and heavy smoking (Anda, Croft et al. 1999). In another retrospective study on four birth cohorts in the USA, the number of childhood adversities increased the risk of smoking and had a consistent, strong and graded relationship with it (Dube, Felitti et al. 2003). Furthermore, it has been found that adolescent smoking is associated with childhood family structure, family environment and attachment to family (Tyas and Pederson 1998).

Smoking in one's primary social environment, parental smoking (Green, Macintyre et al. 1991; Rossow and Rise 1994; White, Pandina et al. 2002; White, Hopper et al. 2003; Barman, Pulkkinen et al. 2004; Fagan, Brook et al. 2005; Brook, Pahl et al. 2006), especially a smoking mother (Kandel, Wu et al. 1994; Kandel 1995), peer smoking (West, Sweeting et al. 1999; White, Pandina et al. 2002; White, Hopper et al. 2003; Brook, Pahl et al. 2006) and smoking siblings (Slomkowski, Rende et al. 2005) have been found to predict smoking, although findings on the determinants are partly inconsistent (Avenevoli and Merikangas 2003) and gender-specific (White, Pandina et al. 2002). A review of 87 studies on familial influences on adolescent smoking revealed that findings across the studies show weak and inconsistent associations between parental and adolescent smoking. The underlying reason for this was thought to lie in methodological issues and associated factors (Avenevoli and Merikangas 2003). Maternal smoking appears to have a greater impact on children's smoking than paternal smoking. Women's smoking behaviour affects the process of childhood socialisation into smoking, and mother's smoking attitudes and practices seem to have a strong influence on children's smoking behaviour (Graham 1987). Maternal smoking can affect offspring even before birth (Jaddoe, Troe et al. 2008). Nicotine and other substances released by maternal smoking can affect the foetus, perhaps through nicotinic input to the dopaminergic motivational system (Kandel, Wu et al. 1994). It is



notable that the effect of parental smoking on offspring smoking may be confounded by parental education as parents from lower SEP also tend to smoke more.

Smoking behaviour is also influenced by genetic factors (White, Hopper et al. 2003). It has been found that different aspects of smoking behaviour, such as age of initiation, quantity of smoking and smoking cessation are partly influenced by the same genetic component, although part of the genetic influence is different (Broms, Silventoinen et al. 2006). Many years of twin and adoption studies have demonstrated that heritability is at least 50% responsible for both smoking initiation and smoking persistence. Furthermore, the extent to which genetic and environmental factors contribute to smoking behaviour in men is significantly different from that in women (Li 2003). Smoking initiation has been found to be influenced by genetic factors and shared environmental influences. According to some findings, once smoking is initiated, genetic factors determine to a larger extent the quantity that is smoked (Koopmans, Slutske et al. 1999). Other studies have found no differences between men and women in the magnitude of genetic and environmental influences on individual differences in smoking initiation and quantities smoked. Environmental factors play the greatest role in determining variation in tobacco smoking in adolescents and young adults. However, genes also seem to have a direct influence on variation in young adults' smoking behaviours (White, Hopper et al. 2003). Twin studies have also demonstrated that the importance of genetic and environmental influences varies across the development for substance use (Dick, Pagan et al. 2007).

#### Adult risk factors and potential pathways to adult smoking

A follow-up study based on a cohort followed from age 16 to 30 concluded that adult smoking reflects the cumulative influence of multiple socioeconomic and psychosocial chains of risks experienced during upbringing (Novak, Ahlgren et al. 2007). Stressful childhood experiences and their relation with later determinants of smoking is still not well known (Avenevoli and Merikangas 2003), although numerous potential adult risk factors for daily smoking have emerged from earlier epidemiological research. Age, gender and socioeconomic factors (Marsh and McKay 1994; van de Mheen, Stronks et al. 1998; Paavola, Vartiainen et al. 2004; Laaksonen, Rahkonen et al. 2005; Power, Graham et al. 2005; Rahkonen, Laaksonen et al. 2005), marital status related factors (Joung, Stronks et al. 1995) and area-level (Diez Roux, Merkin et al. 2003; Giskes, van Lenthe et al. 2006) sociodemographic characteristics and adverse life events are most frequently identified as risk factors for smoking.

Educational attainment is one potential mediating factor in the relationship between childhood circumstances and smoking in adulthood (Lawlor, Batty et al. 2005). In the British birth cohorts, persistent smoking shows strong social gradients with both childhood and adulthood socioeconomic measures. However, in men the association



with childhood circumstances was no longer statistically significant after adjusting for adult circumstances (Jefferis, Power et al. 2004). According to a Finnish follow-up study (TAM), smoking was more prevalent in young adults coming from a manual class of origin than in those from other classes. When the person's own social class was controlled for, the effect of parental social class decreased the differences but they remained statistically significant. This result indicates that parental social position has effects on early adult smoking other than those mediated by current SEP (Huurre, Aro et al. 2003). According to another Finnish data (HHS), smoking is associated with structural, material as well as perceived dimensions of socioeconomic disadvantage in the adult population (Laaksonen, Rahkonen et al. 2005). In general, there are more smokers in lower social classes and in those with a lower education or economic difficulties (Marsh and McKay 1994; Borg and Kristensen 2000; Power, Graham et al. 2005; Rahkonen, Laaksonen et al. 2005), although the effects of SEP on smoking are slightly different in different countries because of differences in the diffusion of smoking (Cavelaars, Kunst et al. 2000). Not only smoking, but also smoking cessation seems to vary according to SEP. A Finnish study based on a large prospective twin dataset suggested that high education predicts smoking cessation in both genders, as does high social class in women. In general, socioeconomic indicators seem to be important predictors of smoking cessation (Broms, Silventoinen et al. 2004).

Employment paths are another potential mediator between childhood circumstances and smoking in young adults. Previous studies have shown that employment status is associated with smoking in adulthood (Marsh and McKay 1994; Borg and Kristensen 2000; Power, Graham et al. 2005; Rahkonen, Laaksonen et al. 2005). The association between long-term unemployment and smoking seems to be even stronger in young people than in adults (Reine, Novo et al. 2004). A French study on the relationship between unemployment and the prevalence of risk behaviour in men indicated that unemployed men have a significantly higher prevalence of smoking than the working population (Khlat, Sermet et al. 2004).

Adult family structure is associated with smoking in adulthood. Having children has been found to be associated with smoking cessation in parents, whether poor or affluent (Jarvis 1996). Smoking in single parents is more common than in parents living together. Single parenthood is associated with smoking in both men and women independent of education, occupational social class, household disposable income, housing tenure or social relations (Rahkonen, Laaksonen et al. 2005). Early motherhood, non-cohabitation and single motherhood increase the odds of smoking in UK women as well (Graham, Francis et al. 2006). In men, marriage has been found to be associated with an increased probability of smoking cessation (Broms, Silventoinen et al. 2004).

As regards area of residence as a risk factor for smoking, a recent study on differences in smoking prevalence between urban and non-urban areas in six Western European countries found that smoking prevalence was highest in urban areas (Idris, Giskes et al. 2007). In a follow-up study of a white population in the USA, there was some evidence of a contextual effect of the area characteristics on smoking: living in the most disadvantaged areas was associated with a significantly higher prevalence of smoking even after controlling for individual socioeconomic characteristics (Diez Roux, Merkin et al. 2003). In another study from the USA, it was suggested that living in a deprived area seems to reduce the likelihood of quitting smoking (Giskes, van Lenthe et al. 2006). Based on the Dutch GLOBE study, residents living in the socioeconomically most disadvantaged neighbourhoods were more likely to smoke than those living in the most advantaged neighbourhoods (adjusted for age, gender, education, occupation and employment status). It was concluded that physical neighbourhood stressors were related to smoking, even over and above individual level characteristics (van Lenthe and Mackenbach 2006).

### 3.2.2 Unhealthy alcohol use

In several countries alcohol consumption is highest among young adults (Fillmore, Hartka et al. 1991; Quigley and Marlatt 1996; Anthony and Echeagaray-Wagner 2000; Casswell, Pledger et al. 2003; Poelen, Scholte et al. 2005). Many of them are heavy drinkers, and studies on the continuity of such behaviour suggest that many also continue this drinking pattern into adulthood (Bennett, McCrady et al. 1999; Paavola, Vartiainen et al. 2004). This is true particularly of those with very high levels of consumption (Pape and Hammer 1996). It has been shown that the frequency of drinking increases in early adult years and that the quantities consumed peak at age 21, decreasing thereafter for both genders (Casswell, Pledger et al. 2003). Heavy drinking is one of the most important pathways to poor health and serious diseases (Dong, Dube et al. 2003; Dube, Felitti et al. 2003), premature mortality (Mäkelä 1999; Pensola 2004) and health inequalities (Power and Matthews 1997; Schrijvers, Stronks et al. 1999). In addition, in young people, the health risks of excessive alcohol consumption include an increased likelihood of involvement in violence and participation in other risky activities (Furlong and Cartmel 2007).

Unhealthy alcohol use, whether measured in terms of excessive alcohol consumption, substance use disorders or alcohol abuse, has been found to be associated with both childhood and adulthood circumstances. Several studies have demonstrated the effect of low parental SEP and adverse childhood circumstances on heavy drinking and alcohol problems in adolescence and adulthood (Droomers, Schrijvers et al. 1999; McArdle, Wiegersma et al. 2002; Droomers, Schrijvers et al. 2003; Zlotnick, Tam et al. 2004; Yang, Lynch et al. 2007). Among the childhood factors and adversities

that have been found to be associated with unhealthy alcohol use (heavy drinking or alcohol disorders) in either or both genders in adulthood, are parental divorce (Kuh and Maclean 1990; Andreasson, Allebeck et al. 1993; Hope, Power et al. 1998; Anda, Whitfield et al. 2002), sexual abuse (Wilsnack, Vogeltanz et al. 1997; Sartor, Lynskey et al. 2007), poor family functioning (Jacob and Johnson 1997; Engels, Vermulst et al. 2005), family economy (Andreasson, Allebeck et al. 1992; Andreasson, Allebeck et al. 1993), parental mental health problems (Anda, Whitfield et al. 2002) and parental alcohol abuse (West and Prinz 1987; Green, Macintyre et al. 1991; Henderson, Albright et al. 1994; Pulkkinen and Pitkänen 1994; Steinhausen 1995; Anda, Whitfield et al. 2002; Pirkola, Isometsä et al. 2005).

### Childhood predictors of unhealthy alcohol use

Low parental SEP predicts heavy alcohol use in adolescence and adulthood. Droomers and her colleagues studied a cohort of New Zealand adolescents aged 11–21 years and found a significant association between paternal occupation and adolescents' alcohol consumption, emerging at age 15. In this study, those from the lowest occupational groups were almost twice as likely to be heavy drinkers as those in the highest occupational group. Based on these findings, it seems that socioeconomic background substantially affects at least adolescent alcohol consumption and contributes to the accumulation of disadvantage (Droomers, Schrijvers et al. 2003). Some other studies, however, have found no association between primary SEP and alcohol use in adolescence (Tuinstra, Groothoff et al. 1998). Evidence on the impact of parental SEP on young adults' alcohol use is scarcer. Childhood family structure is associated with unhealthy alcohol use later in life, although living with both parents seems to be a less robust barrier to substance use than qualitative aspects of family life, particularly attachment to mothers (McArdle, Wieggersma et al. 2002). Factors related to parental support and control continue to be of significance even when the family's SEP is taken into account, and family socialisation factors would appear to be of more direct importance to adolescent drinking behaviour (Shucksmith, Glendinning et al. 1997).

Parental alcohol problems are important risk factors for heavy alcohol use and alcohol use disorders in adulthood. It has been suggested that children of alcohol misusing parents are prone to genetic and environmental risk factors (Steinhausen 1995). In fact, exposure to parental alcohol abuse may be closely associated with experiencing other childhood adversities (Dube, Anda et al. 2001). In the United States, the ACE Study revealed that the risk of several childhood adversities was significantly greater in those reporting parental alcohol abuse. In addition, the number of adverse childhood experiences had a graded relationship to alcoholism in adulthood (Anda, Whitfield et al. 2002). In another study based on the same data, it was suggested that many ACEs were associated with a higher risk of heavy alcohol use as an adult:

compared to persons with no childhood adversities, the risk of heavy drinking and self-reported alcoholism were twofold to fourfold in those with many childhood adversities, even when controlling for parental alcoholism (Dube, Anda et al. 2002). The genetic vulnerability of children of alcoholics increases the risk of substance abuse. In addition, neuropsychological effects of maternal alcohol consumption in pregnancy are common, and paternal alcohol abuse may contribute to foetal damage as well. Family drinking patterns are associated with adolescents' alcohol abuse, and early induction increases the risk of addiction later as well (Zeitlin 1994).

Some work has also been done to explore the long-term consequences of parental divorce on adult alcohol use. In British women, parental divorce has been found to be associated with higher alcohol consumption (Kuh and Maclean 1990). However, there is some indication that this effect is different at different phases of adulthood. A study based on the 1958 British birth cohort followed to age 33 years found that at age 23, the relationship between parental separation and alcohol consumption was weak and inconsistent, but by age 33 stronger and more consistent. High levels of alcohol consumption, heavy drinking and problem drinking were found in those who had experienced parental divorce in childhood, but not later parental divorce. It was concluded that the risk associated with early parental divorce appeared to strengthen between ages 23 and 33 (Hope, Power et al. 1998). In a study of a large representative British sample, it was found that parental loss was not in fact an antecedent to heavy drinking in young adults at all (Estaugh and Power 1991). Regarding other childhood adversities, childhood physical abuse proved to be a strong predictor of current substance abuse in early adulthood, although sexual abuse did not. Depression was shown to mediate the relationship of physical abuse to current alcohol abuse (Lo and Cheng 2007).

However, alcohol use also depends on the age of alcohol initiation and drinking patterns in adolescence (Poikolainen, Tuulio-Henriksson et al. 2001; Andersen, Due et al. 2003; Bonomo, Bowes et al. 2004; Wells, Horwood et al. 2004). A recent US study showed that children with particular adverse childhood experiences may initiate drinking earlier than their peers and be more likely to drink to cope with problems, rather than for pleasure or to be social (Rothman, Edwards et al. 2008). Friends and peer group affect drinking in adolescence as well, and girls have been suggested to be more susceptible to friends' influence (Dick, Pagan et al. 2007).

#### Adult risk factors and potential pathways from childhood to adult alcohol use

Adult characteristics associated with both childhood circumstances and heavy drinking potentially mediate the effect of childhood circumstances on drinking behaviour. Various concurrent individual level determinants including sociodemographic and

psychosocial factors affect unhealthy alcohol use in adulthood (Holman, Jensen et al. 1993; Poikolainen 2005; Pagan, Rose et al. 2006). However, it is not known whether the effect of the early social environment on heavy drinking is mainly independent of these later experiences.

Educational attainment is one significant mediating factor in the relationship between socioeconomic adversity and unhealthy alcohol use (Lawlor, Batty et al. 2005). It has been suggested that the distribution of alcohol consumption by SEP is not universal as the social inequalities differ by gender, indicator of alcohol use and across groups of countries (Bloomfield, Grittner et al. 2006). In general, higher social groups drink more frequently but smaller quantities at a time than lower social groups. Higher income has been found to be associated with a higher frequency of drinking, whereas quantities consumed are usually influenced by educational level. In young adults in New Zealand, the less-educated drink significantly more during one drinking occasion, and those with a higher income drink most frequently (Casswell, Pledger et al. 2003). However, it has been suggested that alcohol consumption is more excessive in higher status groups (Ahlström, Bloomfield et al. 2001), and according to some studies, in lower status groups (Droomers, Schrijvers et al. 1999; van Oers, Bongers et al. 1999; Droomers, Schrijvers et al. 2004; O'Donnell, Wardle et al. 2006). For example, in the USA, those who took academically advanced courses in high school had higher rates of current and binge drinking after high school (aged 20–26 years). This association was explained partly by educational, family and work circumstances in early adulthood (Crosnoe and Riegle-Crumb 2007). As regards the cumulative effect of problems, a Finnish study revealed that an unstable career line in men at age 26 was related to drinking problems; the pathway leading to drinking problems in men included problems in school adjustment and in the family at age 14 (Rönkä and Pulkkinen 1995).

With respect to employment paths, some studies have suggested that unemployment is strongly related to heavy alcohol use (Lee, Crombie et al. 1990; Power and Estaugh 1990; Luoto, Poikolainen et al. 1998; Montgomery, Cook et al. 1998; Khlát, Sermet et al. 2004), particularly in women (Ahlström, Bloomfield et al. 2001), although opposite findings have also been reported (Hammer 1992). Overall, the unemployed have higher levels of alcohol consumption (Luoto, Poikolainen et al. 1998), although in some studies this result is largely influenced by the excess of heavy drinkers (Lee, Crombie et al. 1990). However, early employment experiences do appear to be relevant to the drinking of young adults, since unemployment of six months or more in total has been found to be significantly associated with heavier drinking in men (Power and Estaugh 1990). The effect of unemployment on health and health behaviour varies naturally according to the duration of unemployment as well as the socio-cultural context, for example during periods of high and low unemployment.

As regards family structure and living arrangements, single and divorced persons are more likely to use excessive amounts of alcohol than married persons (Joung, Stronks et al. 1995; Power, Rodgers et al. 1999; Bogart, Collins et al. 2005; Joutsenniemi, Martelin et al. 2007). Childlessness (Ahlström, Bloomfield et al. 2001) and living in urban areas (Sundquist and Frank 2004) have also been found to be risk factors for heavy alcohol use. Psychosocial factors are possible mediating factors between childhood circumstances and later heavy alcohol use. For example, depression has been shown to mediate the relationship between physical abuse in childhood and later alcohol abuse (Lo and Cheng 2007). Depressive symptoms and other psychiatric disorders, are often associated with problematic alcohol use (Haarasilta, Marttunen et al. 2004; Buckner, Keough et al. 2006).

### 3.2.3 Overweight and obesity

The prevalence of obesity has increased dramatically in the past few decades, particularly in Western countries (WHO 2000; James 2004; Mizuno, Shu et al. 2004; Seidell 2005). The problem has spread to ever younger segments of the population (Livingstone 2000; Ebbeling, Pawlak et al. 2002), and the prevalence of obesity has increased markedly in young adults as well (Kark and Rasmussen 2005; Mohler-Kuo, Wydler et al. 2006; Lahti-Koski, Harald et al. 2007; Svensson, Reas et al. 2007). In young Swedish men, the prevalence of overweight and obesity has steadily increased over the past three decades. For example, during 1970–2000, mean BMI has increased from 20.89 kg/m<sup>2</sup> to 22.49 kg/m<sup>2</sup>, while the prevalence of overweight increased twofold and the prevalence of obesity fourfold. Another noteworthy finding was the discovery that the socioeconomic gap in obesity had increased over time (Kark and Rasmussen 2005). In Finland, a similar pattern can be seen in the development of weight gain in young men (Santtila, Kyröläinen et al. 2006; Lahti-Koski, Harald et al. 2007).

The detrimental health consequences of obesity are well documented. Among other health risks, it contributes to a higher incidence of cardiovascular diseases, type 2 diabetes mellitus, hypertension, infertility and certain cancers as well as functional impairment (Rissanen, Heliövaara et al. 1990; James 1998; WHO 2000), and is associated with long-term sickness absence (Moreau, Valente et al. 2004; Laaksonen, Piha et al. 2007) as well as retirement on disability pension (Rissanen, Heliövaara et al. 1990; Karnehed, Rasmussen et al. 2006). A Finnish study on employees of the City of Helsinki (HHS) revealed that obesity increases the risk of short and long periods of sickness absence in adults (Laaksonen, Piha et al. 2007). Obesity also has severe psychosocial, social and economic consequences (Rissanen 1996; Laitinen, Power et al. 2002).

Obesity is increasingly apparent even in childhood and youth and is highly prevalent in adulthood. It is therefore one of the main public health issues and has prompted increasing research on its determinants (Lawlor and Chaturvedi 2006). In general, it has been suggested that obesity and overweight are largely a consequence of over-nutrition and under-activity (Lawlor and Chaturvedi 2006). However, it seems that genetic, environmental and other behavioural factors as well as their interaction are also at play (Parsons, Power et al. 1999). For instance, prenatal factors and high birth weight, parental fatness, early maturation and several social, behavioural and psychological factors have been found to be associated with obesity in adulthood (Parsons, Power et al. 1999; Power and Parsons 2000; Ball, Mishra et al. 2003; James 2004). Some findings suggest that exposure to unfavourable circumstances in childhood is associated with excess weight and weight gain in adulthood (Giskes, van Lenthe et al. 2008). However, despite an extensive literature, there is to date only modest evidence for most of the factors that are suspected to play a role in the development of obesity (Parsons, Power et al. 1999).

#### Childhood predictors of overweight and obesity

The findings concerning the relationship between childhood socioeconomic position (SEP) and obesity in childhood are not consistent (Huerta, Bibi et al. 2006). However, there is strong evidence on the effects of low primary social background on adult overweight and obesity (Power and Moynihan 1988; Kark and Rasmussen 2005; Power, Graham et al. 2005; Crossman, Sullivan et al. 2006). Factors related to disadvantaged social origins appear to increase the risk of obesity, particularly in women in Western countries (Power, Graham et al. 2005). In Finland (TAM), women from a manual class of origin have higher rates of overweight and higher BMI than those in other groups (Huurre, Aro et al. 2003). In young adult Swedish men, mean BMI and the prevalence of overweight and obesity was higher in those with low-educated mothers than in those with high-educated mothers (Kark and Rasmussen 2005), whereas father's occupation has been shown to be associated with BMI in Scottish men (Blane, Hart et al. 1996). It is notable that even if an association does exist between one's primary SEP and obesity, it may be confounded by parental obesity (Wada and Ueda 1990), which too few studies have been able to take into account (Parsons, Power et al. 1999). A study from northwest Germany revealed that overweight families of low SEP have a higher risk of overweight and obese children (Danielzik, Czerwinski-Mast et al. 2004). Parental overweight, low SEP and high birth weight are the major determinants of overweight and obesity in childhood (Danielzik, Czerwinski-Mast et al. 2004; Dubois and Girard 2006) and later in life (Whitaker, Wright et al. 1997). However, both familial environmental and genetic factors contribute to the relationship of low parental SEP to adult fatness (Teasdale, Sørensen et al. 1990). The relative contributions of genes and inherited lifestyle factors



to the parent-child fatness association remain largely unknown (Parsons, Power et al. 1999). The influence of other social factors such as family size, number of parents at home and child care has received only limited research attention (Parsons, Power et al. 1999).

Several childhood social circumstances have been found to be associated with later overweight and obesity (Johnson, Cohen et al. 2002; Bachman 2004; Thomas, Hyppönen et al. 2008). Experienced in childhood, parental neglect (Lissau and Sørensen 1994), self-reported sexual and non-sexual abuse (Felitti 1993; Williamson, Thompson et al. 2002), parental alcoholism (Felitti 1993), school difficulties (Lissau and Sørensen 1993) and rearing area (Lissau and Sørensen 1992) have been found to increase the risk of obesity, for example. Based on results from the 1958 British birth cohort, the risk of obesity increased by 20–50% for several adversities (physical abuse, verbal abuse, witnessed abuse, humiliation, neglect, strict upbringing, physical punishment, conflict or tension, low parental aspirations or interest in education, infrequent outings with parents, and father hardly reads to child) (Thomas, Hyppönen et al. 2008). The risk of being obese in early adulthood is also found to be increased if the child had learning difficulties, scholastic proficiency below the class average or scholarly difficulties (Lissau and Sørensen 1993). Childhood overweight has also been found to be associated with severe obesity in both women and men, the association being stronger in men, which demonstrates the importance of childhood overweight as a risk factor for severe obesity over the life-course (Whitaker, Wright et al. 1997; Williams, Davie et al. 1999; Ferraro, Thorpe et al. 2003). Other studies have also emphasised the association between childhood overweight and later obesity: high normal weight status in childhood predicts overweight or obesity in adulthood (Field, Cook et al. 2005).

#### Adult risk factors and potential pathways to overweight and obesity

There are several potential pathways from childhood social circumstances to adult overweight and obesity (Parsons, Power et al. 1999). For instance, childhood circumstances can influence educational career, which in turn affects SEP and health behaviours in adulthood, leading further to obesity. Furthermore, the childhood environment itself may have long-term impact on obesity through, for example, nutrition in infancy (undernutrition, overnutrition) (Power and Parsons 2000), psychological and social factors (e.g. emotional deprivation) (Kaplan and Kaplan 1957; French, Story et al. 1995; French, Perry et al. 1996) and social and cultural norms (attitudes and restraints) (Jeffery, French et al. 1991; Wardle, Volz et al. 1995; Jeffery 1996; Jeffery and French 1996). There is a scarcity of research into the complex relationships between childhood circumstances and adult obesity (Parsons, Power et al. 1999; Power and Parsons 2000).



A recent study on the British 1958 birth cohort investigated how different stressful emotional or neglectful childhood adversities were related to adiposity in mid-adulthood, taking into account childhood circumstances, and whether the relationships were mediated by adult health behaviours and socioeconomic position. According to the results, some severe forms of childhood adversity, such as physical abuse or witnessing abuse of a family member, were associated with an increased risk of obesity and were not fully explained by confounding from other childhood influences or mediation by adult socioeconomic or lifestyle factors. However, some less severe stressful emotional environments showed moderate-to-weak effects, and these appeared to be largely explained by other childhood circumstances (Thomas, Hyppönen et al. 2008).

Educational attainment is an important potential mediating factor in the relationship between socioeconomic adversity in childhood and overweight in adulthood (Lawlor, Batty et al. 2005). However, some studies suggest that primary SEP has independent effects as well (Giskes, van Lenthe et al. 2008). In Finland, women from a manual class of origin have higher rates of overweight and higher BMI than those in other groups. After controlling for the person's own SEP, the effect of parental SEP diminishes but remains significant in women up to 32 years of age (Huurre, Aro et al. 2003). In general, education is a strong determinant of obesity in Finland, especially in women, although the social gradient in BMI has not widened in the 1990s (Lahti-Koski, Vartiainen et al. 2000). The effects of primary and current SEP may be gender-specific. Based on the Dutch GLOBE study, some findings have shown that in adult women, childhood SEP has a greater effect on weight than SEP in adulthood. However, adult SEP seems to have a greater impact than childhood circumstances on weight in men (Giskes, van Lenthe et al. 2008). In addition, factors related to disadvantaged social origins appear to increase the risk of obesity, particularly in women (Power, Graham et al. 2005). Socioeconomic differences in the prevalence of overweight and obesity have been found to be negligible in childhood but marked by early adulthood, with the percentage of overweight and obesity being higher in the lower social classes. SEP and obesity have shown a strong inverse relationship in women in developed societies, but for men and children this relationship has been rather inconsistent (Sobal and Stunkard 1989). The predominant result of a review of 333 studies published during 1988–2004 was the inverse association between SEP and obesity in women (McLaren 2007). In men, obesity has been positively associated with income and in both genders negatively with education (Ward, Tarasuk et al. 2007). It has been suggested that social inequalities in overweight reflect the cumulative influence of multiple adverse circumstances experienced from adolescence to early adulthood (Novak, Ahlgren et al. 2006). However, childhood overweight can affect later social conditions: childhood overweight and obesity may cause selection into more unfavourable tracks of education, for example. It has been found that overweight and obesity at age 14 are

associated with a low level of education, and with being single or divorced at 31 years of age in women (Laitinen, Power et al. 2002).

Part of the protection against weight gain in higher SEP groups may come through the higher frequency of weight monitoring, a lower threshold to define oneself overweight, and a greater likelihood of efforts to weight control (Wardle and Griffith 2001). Social attitudes towards obesity and thinness also vary, and several variables may mediate the influence of attitudes towards obesity that result in the inverse relationship. These include dietary restraint, physical activity and inheritance (Sobal and Stunkard 1989). It has also been suggested that childhood adversities may contribute to a greater risk of eating disorders and problems with eating and weight that may persist into early adulthood (Johnson, Cohen et al. 2002).

Employment paths may also mediate the effect of childhood circumstances on adult obesity, as employment status seems to be associated with overweight and obesity. However, a study based on the 1966 Northern Finland birth cohort found that a long history of unemployment was associated with an increased risk of obesity in women, but not in men, after controlling for potential confounding factors (social class at 14 yrs, BMI at 14 yrs, school performance at 16 yrs, place of residence, and number of children) (Laitinen, Power et al. 2002). In women in Finland, overweight has also been found to be associated with current unemployment and obesity with long-term unemployment and with low individual earnings. Obese women are also most likely to have low household disposable and individual incomes. It seems that deviant body weight is associated with social and economic disadvantage in a gender-specific way and that obese women face multiple social and economic disadvantages (Sarlio-Lähteenkorva and Lahelma 1999). It has been found that non-married women in Denmark are more likely to be obese than their married counterparts, whereas in Finland no such associations have been found (Sarlio-Lähteenkorva, Lissau et al. 2006).

### **3.3 Development of health inequalities in the life-course**

Socioeconomic health inequalities have been observed for decades in many West-European countries, and the general pattern of better health in those with higher SEP is well known (Fox 1989; Rahkonen, Arber et al. 1995; Lahelma and Rahkonen 1997; Davey Smith, Hart et al. 1998; Lahelma, Kivelä et al. 2002; Mackenbach, Bos et al. 2003; Kunst, Bos et al. 2005; Mackenbach, Stirbu et al. 2008). Socioeconomic health inequalities seem to emerge rapidly when heading into adulthood, as they are still very small or non-existent in adolescence (Pensola and Valkonen 2000; Siahpush and Singh 2000; West and Sweeting 2004; Hagquist 2007), but very marked at early middle age (Mackenbach, Kunst et al. 1997; Valkonen, Martikainen et al. 2000; Pensola and

Valkonen 2002). In addition, some studies have argued that the relationship between SEP and health intensifies with age (Rahkonen, Arber et al. 1995; Prus 2004).

There seems to be a somewhat specific period of “equalisation in youth” or “reduction of inequalities” after which health disparities start to increase. Based on several analyses focused on young people in the UK, Patrick West has concluded that despite the correlations between social class and a range of measures of ill-health in childhood and adulthood, early youth is a period of relative equity (West 1988; West, Macintyre et al. 1990; West 1996; West 1997). However, this hypothesis has been challenged from at least two points of view. First, although differences in health measures are small or non-existent, there are differences in health-related behaviours in this age group that have important implications for future health. It has been suggested that socioeconomic differences in health behaviours begin to appear during adolescence when those from lower socioeconomic backgrounds are more often engaged in unhealthy behaviours, such as alcohol consumption, than their peers from higher socioeconomic groups (Droomers, Schrijvers et al. 2003). The same pattern can be seen in smoking habits, diets and alcohol consumption (Lowry, Kann et al. 1996; Roos, Hirvonen et al. 2001). On the other hand, social mobility and educational career have been found to be associated with health-related behavioural patterns during adolescence (Karvonen, Rimpelä et al. 1999; Koivusilta 2000). Secondly, Torsheim and his colleagues have argued that the lack of inequality in West’s analyses may primarily be due to methodological problems (the validity of young people’s reports of parental class), and that although adolescence can be characterised as a period of “reduction in inequalities”, differences in SRH according to material deprivation can be found even in youth in several countries (Torsheim, Currie et al. 2004). On the other hand, it has been suggested that adolescents’ personal social position should be included in studies of health inequalities (Koivusilta, Rimpelä et al. 2006). West reported only few SEP differences in health in youth, a pattern contrasting with that of health inequalities in childhood and adulthood, which may reflect the increasingly pervasive influence of youth culture (West and Sweeting 2004). Social class differentials seem to emerge quite dramatically after this phase of relative equalisation (West 1988).

SEP differences have been explained by reference to mechanisms of causation, selection and their variations (see Chapter 2.2). Following the hypothesis of causation mechanisms, high SEP has been found to promote better living and healthier working conditions (Schrijvers, van de Mheen et al. 1998; Borg and Kristensen 2000; Monden 2005), as well as healthier lifestyle, attitudes and choices (Wardle and Steptoe 2003). Higher SEP has also been found to be associated with physically less strenuous and psychosocially more rewarding work and better housing conditions than lower SEP. Moreover, compared with persons with low SEP, those with high SEP have been found to have less health-damaging behaviours overall. They tend to smoke less (Paavola,

Vartiainen et al. 2004; Laaksonen, Rahkonen et al. 2005; Power, Graham et al. 2005), drink less alcohol (Droomers, Schrijvers et al. 1999; Casswell, Pledger et al. 2003), be physically more active (Lindström, Hanson et al. 2001; Martinez-Gonzalez, Varo et al. 2001), have healthier nutrition habits (Ball, Crawford et al. 2006; Roos, Talala et al. 2008) and are less likely to be obese (Sobal and Stunkard 1989; Ali and Lindström 2006).

However, health itself can have an influence on SEP. Through selection mechanisms, those with poorer health (Haas 2006) and health-damaging lifestyles (Koivusilta, Rimpelä et al. 1998; Koivusilta, Rimpelä et al. 1999; Koivusilta, Rimpelä et al. 2001) may end up in low educational tracks and lower SEP in adulthood. A study based on the British 1958 birth cohort reported that people with poor health were more likely to move down and less likely to move up in the social scale. However, it was concluded that health selection had only a modest effect on the social gradient, and it was not regarded as a major explanation for health inequalities in early adulthood (Manor, Matthews et al. 2003). It has been found that in adolescence, indirect selection based on health behaviours, rather than direct selection by perceived health, contributes to the production of socioeconomic health differences later in life (Koivusilta, Rimpelä et al. 2003). Some studies suggest, however, that the relationship between adult SEP and health outcomes is due mainly to selection effects rather than a causal effect of SEP exposures on health and behaviour (Osler, McGue et al. 2007). A Finnish study revealed that in both genders, the path from psychosomatic symptoms in adolescence to lower education in early adulthood was particularly strong (Huurre, Rahkonen et al. 2005).

Differences in health by SEP may arise from circumstances and experiences in early life that affect one's education, living conditions, health behaviour and health. Socioeconomic health differences may partly be explained by the childhood environment if it is associated with both adult SEP and health in adulthood. For example, social environment in childhood is associated with youth paths (educational career, family formation and employment paths) (Pensola 2004) as well as with health behaviours such as smoking (Anda, Croft et al. 1999; Jefferis, Power et al. 2004), heavy alcohol use (Anda, Whitfield et al. 2002; Engels, Vermulst et al. 2005), obesity (Parsons, Power et al. 1999) and physical activity (Huurre, Aro et al. 2003). As childhood circumstances are also determinants of health (Rahkonen, Lahelma et al. 1997; Dube, Felitti et al. 2003), they may provide a partial explanation for the association between SEP and health. There is some evidence that childhood environment explains part of the SEP differences observed in young adult health. Based on the data collected in the British 1958 birth cohort, SEP differences in health at age 23 were not eliminated after controlling for earlier circumstances. However, substantial reductions in health were associated with a number of factors in childhood, in particular

primary SEP, housing tenure, crowding, family size and receipt of free school meals (Power 1991). In another study, explanations for health inequality at age 33 years spanned from early life to early adulthood (Power, Matthews et al. 1998). It has also been suggested that predictors of cardiovascular disease measured in childhood and adolescence may explain a substantial part of the social gradient in cardiovascular morbidity and mortality in adulthood (Hemmingsson and Lundberg 2005).

The effect of parental home on mortality differences has been found to be mediated through its effect on youth paths. In a Finnish study, however, educational, marital and employment paths had a substantial effect, independent of parental home, on social class differences from various causes of death. Social class differences in total mortality in men in their middle adulthood were only partly determined by parental home, but they were mainly attributable to these youth paths (Pensola and Martikainen 2004). Gradients in psychological distress also reflect the cumulative effect of multiple adversities experienced from childhood to adulthood (Power, Stansfeld et al. 2002). Conditions present or established in youth and adolescence are also of major importance to understand the strong SEP gradient in disability pensions in young men, as the increased risks for skilled and unskilled manual workers compared with non-manual employees might be interpreted according to the concept of unfavourable life careers (Upmark, Lundberg et al. 2001).

Behavioural factors also play a role in the development of health inequalities, although some studies suggest that the higher prevalence of major health-risk behaviours in lower socioeconomic groups is not the dominant mediating mechanism that can explain socioeconomic disparities in physical functioning and self-rated health (Lantz, Lynch et al. 2001). In a recent Finnish study though, smoking, vegetable use and physical activity were the most important health behaviours explaining educational differences in all mortality outcomes, while relative weight and alcohol use were less relevant (Laaksonen, Talala et al. 2008). In a Dutch study, the association between educational level and mortality was largely explained by material factors (financial problems, employment status, income) and to a lesser extent by behavioural factors (alcohol, smoking, body mass index, physical activity) (Schrijvers, Stronks et al. 1999). It has been found that smoking habits at 16 years of age explain more of the variation in self-rated health in early adulthood than health status at 16 years (Power, Manor et al. 1990).

Some studies emphasise the importance of the psychosocial approach to explaining health inequalities and suggest that psychosocial pathways associated with relative disadvantage further reinforce the direct effects of absolute material living standards (e.g. Marmot and Wilkinson 2001; Wilkinson 1996; Marmot and Bobak 2000). Exposure to an adverse psychosocial environment may cause sustained stress reactions with negative long-term consequences for health. Such exposures may be implicated

in the association of socioeconomic status with health (Siegrist and Marmot 2004). According to some results, health is influenced by both material and psychosocial aspects of socioeconomic factors (Pikhart, Bobak et al. 2003). In addition, some findings suggest that stressors may be an important mechanism underlying the social gradient in health (Orpana, Lemyre et al. 2007), others make the strong argument that psychosocial factors have the potential to reduce the burden of ill health and to diminish the social gradient in morbidity and mortality, at least in working people (Marmot 1999). However, it has been argued that a psychosocial interpretation of health inequalities, in terms of perceptions of relative disadvantage and the psychological consequences of inequality, raises several conceptual and empirical problems (Lynch et al 2000). Considerable research has been devoted to the quality of the impact of social relations on health and health inequalities (e.g. Seeman 1996; Oakley 1992). The basic argument has been that social integration reduces the risk of morbidity and mortality and leads to better mental health (Berkman 1995).

In general, it seems that work-related factors are important determinants of health inequalities in the adult population (Hemström 2005). Lundberg suggested in the early 1990s that a large part of the social class differences in physical and mental illness can be understood as a result of differences in living conditions and primarily in differences in working conditions (Lundberg 1991). He showed that physical working conditions are the primary source of class inequality in physical illness, although economic hardship during upbringing and health-related behaviours also contribute (Lundberg 1991; Lundberg 1993). Physical work load (Aittomäki, Lahelma et al. 2007) as well as qualitative aspects of work contribute to health inequalities. For example, a substantial part of the relationship between SEP and health could be attributed to job control whereas job demands reinforce the relation (Rahkonen, Laaksonen et al. 2006). In young adults, too, job characteristics have been found to have a mediating effect in the association between SEP and health. A study on a large sample of Canadian workers aged 20–29 years indicated that job characteristics partly explained the educational gradient observed in work-related injuries and to a lesser extent in self-perceived health. Lifestyle factors, however, had a closer association with the gradient (Karmakar and Breslin 2008). The duration of work exposures also has an effect on health inequalities. In a Dutch population, lifetime exposure to adverse working conditions explained a significant part of the health differences observed between the highest and lowest educated men. In women, only relative lifetime exposure to adverse working conditions explained a small part of the educational differences in health, while current and absolute lifetime exposure did not explain the differences. Measurements of lifetime exposure to working conditions may offer a better explanation for educational differences in health than measurements of current exposure (Monden 2005).

However, it is likely that the development of health inequalities is influenced by several factors and mechanisms over the life-course. In a Dutch study on the general population aged 15–74 years, material factors, psychosocial factors and behavioural factors together reduced the relative risk of mortality, material factors contributing the most to educational inequalities. Part of the contribution of material factors was mediated through psychosocial and behavioural factors, and psychosocial factors contributed to educational inequalities partly via behavioural factors. Behavioural and psychosocial factors contributed only marginally to the explanation independent of material factors (van Oort, van Lenthe et al. 2005).

### **3.4 Summary of the literature review**

The origins of poor adult health and health differences lie in circumstances preceding current social position and living conditions. The theory of social pathways between childhood and adult health emphasises the social chains of risk that operate throughout one's life course (Kuh and Ben-Shlomo 2004). The effect of primary social environment on adult health may be direct, or early living conditions and environments may affect the pathways leading to adult positions. These paths may mediate effects of early circumstances on health, but at the same time they may modify them. An adult risk factor or exposure may mediate the association between childhood exposure and health outcome when it chronologically follows the exposure and is conceptualised as lying, at least partly, on the causal pathway. When entering adulthood, youth paths (educational, employment and family formation paths) may mediate the effects of primary social environment on early adult health, health behaviour and health inequalities.

The research literature gives some indication that parental SEP, other childhood living conditions and adversities have an effect on different aspects of adult health and health behaviour. In general, persons who have lived in adverse economic and social childhood conditions tend to have poorer health (self-rated health, mental health, morbidity and mortality) and more health-damaging behaviour (smoking, unhealthy alcohol use and overweight) in adulthood. However, there is still a lot to know on the importance of living conditions in childhood and youth to adult health, health behaviour and health inequalities, and even less is known about how environmental and behavioural factors mediate the effects of earlier adverse experiences. Much work still needs to be done to explore the combined effects of several childhood living conditions and adversities with current circumstances as determinants of different health outcomes and behavioural patterns in young adults. Moreover, the mechanisms lying behind socioeconomic differences in health are not yet fully understood, despite an abundance of various models based on theories of causation, selection and their modifications.



## 4 AIMS AND FRAMEWORK OF THE STUDY

The main objective of this study is to identify which childhood and contemporary circumstances determine health, health behaviour and health inequalities in young adults (18–29 years) in Finland. In addition, the purpose is to explore the potential mediating factors through which childhood circumstances have an effect on young adult health, health behaviour and educational health differences and to assess the possible pathways from childhood to adult health outcomes.

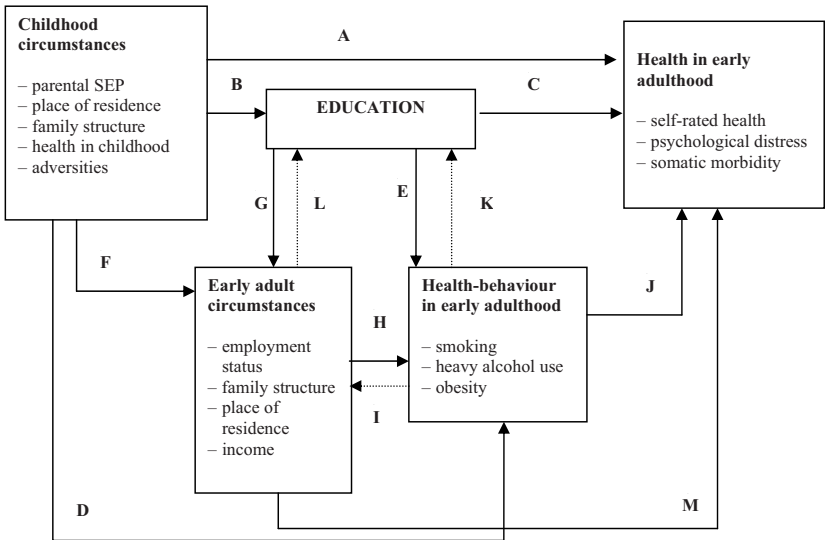
In more detail, the specific aims of this study are to

1. study the role of different aspects of childhood living conditions and adversities as determinants of poor self-rated health, psychological distress and somatic morbidity in early adulthood, and to examine the role of the respondent's own education as a potential mediator in these associations (Substudy I);
2. study the association of childhood living conditions and adversities with three central behavioural risk factors (smoking, heavy drinking and overweight/obesity) and to examine the role of the respondent's own education and other current circumstances in these associations (Substudies II–IV); and to
3. determine to what extent childhood social circumstances, current circumstances and indicators of health behaviour explain the educational differences in self-rated health, and to what extent the effect of childhood circumstances on educational health differences in early adulthood is shared with the effects of later health behaviour and current living conditions (Substudy V).

The simplified model (Figure 1) of the key associations between the factors analysed in this study is based on the life-course approach and the model of social pathways presented earlier. It outlines the framework of this study and presents the main associations as operationalised in this study. Most of these associations are to a lesser or greater extent reciprocal, and some factors may also share other connections with one another. For the sake of clarity, those connections that are not the focus of this study do not appear in the simplified model.



**Figure 1.** *A simplified model of the associations between childhood circumstances, education and other adult circumstances, health behaviour and health as operationalised in this study.*



In the first Substudy (I) the aim is to explore the association between childhood circumstances and health in early adulthood (A) and to establish whether this association is mediated through the respondent's own education (B+C). Studies II–IV assess the associations between childhood circumstances and health behaviour indicators (D) and examine whether this association is mediated through the respondent's own educational level (B+E), other early adult circumstances (F+H) or the sequence of these (B+G+H). However, it is possible that health-damaging behaviour adopted in adolescence affect the early adult circumstances and education as well (I and K).

The fifth Substudy (V) examines which factors contribute to the association between education and health (C). Health behaviours may mediate the association between education and health: education may promote healthier behavioural patterns (E), which in turn affect health (J). However, the opposite causal order between education and health behaviours is also plausible. Health-damaging behaviours adopted early in adolescence may partly select people to different educational positions (K), and thus explain part of the educational health differences. In the same way, early adult living conditions associated with both education (G and L) and health (M) may explain or mediate part of the association between education and health. Childhood circumstances

are taken into account as potential explanatory factors potentially affecting both the respondent's education (B) and health (A). As childhood circumstances are assumed to affect health behaviour (D) and living conditions in early adulthood (F), part of the contribution of childhood circumstances to educational health differences may be shared with that of the latter two categories of factors.

#### Direct and mediated effects

The focus in this thesis is on the direct and mediated effects of childhood living conditions on early adult health and health behaviour. Once the relationship between two variables is established, the effect of a third variable on this association is often considered (Valkonen and Martelin 1988; MacKinnon, Krull et al. 2000). One possible reason for doing so is a potential causal process in which the independent variable (here childhood factors) affects the dependent variable (health or health behavioural outcome). This may be referred to as a mediational hypothesis (James and Brett 1984). The hypothesis comprises two causal paths: the first one links the independent variable to the dependent variable directly (*the direct effect*), and the other one links the independent variable to the dependent variable through the mediator (*the mediated effect*). This indirect or mediated effect means that the independent variable causes the mediator, which in turn causes the dependent variable. In the mediational context the relationship between two variables is reduced because the mediator explains a major part of the association, because it is on the causal path between these two variables (MacKinnon, Krull et al. 2000). The mediator can be characterised as a factor associated with both exposure and outcome (in this study childhood factors and health outcome), but it does not occur prior to or simultaneously with the exposure.

It is important to distinguish mediation from confounding effects. A possible confounder is also associated with exposure and with outcome, but it is not a consequence of the exposure for it occurs temporally prior to or simultaneously with the exposure. Possible confounding factors related to both exposure and the outcome of interest may falsely obscure or accentuate the relationship between them (MacKinnon, Krull et al. 2000).

It is also possible that the third factor between exposure and outcome (e.g. education) modifies the relationship between them. This would mean, for example, that the effect of childhood circumstances on early adult health is different in different educational categories. However, because of the limitations of the data used in this thesis, the effect of modification is beyond the scope of this study.

## 5 DATA AND METHODS

### 5.1 Study design and participants

The data for this study were collected in 2000–2001 as part of the Health 2000 Survey (N=9,922), a cross-sectional nationwide health interview and examination survey (Aromaa and Koskinen 2004). Two subsets of the nationally representative data are used in this thesis: the first comprised the age group 18–29 (N=1,894) (Substudies I–V), and the other one the age group 30 years and over (N=8,028), in which the data for those aged 30–39 (N=1,775) were used (Substudy I). Both datasets include information collected by structured computer-aided interviews (CAPI) and self-administered questionnaires.

In the age group 18–29 years, 1,505 young adults participated in the interview phase (79% of the original sample). The questionnaire was returned by 1,282 persons, i.e. 85% of the interviewed participants. In the age group 30–39 years, 1,529 persons took part in the interview (86%), and 1,448 completed and returned the questionnaire (95% of the interviewed participants). Specific participation rates are presented in Table 1.

**Table 1.** *Main outcomes, age ranges, participants and data inclusion criteria in original studies (I–V) of the cross-sectional Health 2000 Survey*

Study	Outcome	Age	Participants	Inclusion criteria
I	Poor SRH, somatic morbidity, psychological distress	18–39	Sample: N=3,669, (1,845 men, 1,824 women) Participants: n= 3,049 (1,498 men, 1,551 women) Participation rate: 83%	All
II	Daily smoking	18–29	Sample: N=1,894 (981 men, 913 women) Participants n= 1,505 (765 men, 740 women) Participation rate: 79% Met inclusion criteria: n=1,282 (602 men, 680 women) Final participation rate: 68%	Questionnaire completed
III	Heavy drinking	18–29	Sample: N=1,894 (981 men, 913 women) Participants n= 1,505 (765 men, 740 women) Participation rate: 79% Met inclusion criteria: n=1,234 (602 men, 632 women) Final participation rate: 65%	Not pregnant Questionnaire completed
IV	Overweight and obesity	18–29	Sample: N=1,894, 981 men, 913 women, Participants n= 1,505, 765 men, 740 women Participation rate: 79% Met inclusion criteria: n=1,158 (590 men, 568 women) Final participation rate: 61%	Questionnaire completed Not pregnant Not given birth 0–6 months before data collection Not underweight Weight and height information
V	Educational differences in poor self-rated health	18–29	Sample: N=1,894 (981 men, 913 women) Participants n= 1,505 (765 men, 740 women) Participation rate: 79% Met inclusion criteria: n=1,282 (602 men, 680 women) Final participation rate: 68%	Questionnaire completed

**Figure 2. The study areas of the Health 2000 Survey ([www.terveys2000.fi](http://www.terveys2000.fi)). The boundaries of the university hospital districts are marked in bold line and the participating municipalities are shown in color on the map.**



The data are representative of the entire country. Data collection used a two-stage cluster sampling design: in the first stage the 15 largest and 65 other health centre districts were selected as the primary sampling units, and in the second stage persons were randomly selected from these districts. More specifically, the frame was regionally stratified according to the country's five university hospital districts (Figure 2), each of which comprises about one million inhabitants. From each university hospital districts, 16 health care districts were sampled as clusters. The 15 largest cities were all included in the sample with the probability of one, and the remaining 65 health care districts were selected by systematic PPS sampling (probability proportional to size). Data collection, participation rates at different stages of the survey, non-participation as well as the sampling design and selection process for the Health 2000 Survey are described in more detail elsewhere (Aromaa and Koskinen 2004; Laiho and Nieminen 2004; Heistaro 2005; Koskinen, Kestilä et al. 2005; Laiho 2006; Koskinen, Laiho et al. 2008).

The study design is cross-sectional and includes retrospective inquiries on the respondents' childhood and youth. Although retrospective studies based on recalled information may yield less reliable information about the explanatory variables than prospective studies do, they nevertheless have the power to establish the causal order of predictors and outcomes. The Health 2000 data provided a useful basis for identifying operational counterparts for most of the key concepts presented in the framework

of this thesis (Figure 1). For example, childhood environment is characterised by socioeconomic position (SEP) and economic and social problems experienced during childhood and adolescence. Educational career, adult circumstances, health behaviour and various health measures are comprehensively described as well.

## 5.2 Study variables and definitions

Table 2 summarises the variables used in the original studies and their literature reference, if available. The variables are described and defined in more detail in the original publications (I–V).

### 5.2.1 Indicators of health

*Self-rated health (SRH)* (I and V) is a widely used global measure of health, which in this thesis was measured by the question, “In general, would you say your health is...”, with five response options ranging from good to poor. Participants reporting the three poorest levels (‘average’, ‘quite poor’, ‘poor’) of health were classified as having “poor SRH”. SRH is an important tool in studying a population’s health (Krause and Jay 1994; Heistaro, Vartiainen et al. 1996; Martikainen, Aromaa et al. 1999; Eriksson, Undén et al. 2001; Manderbacka, Lahelma et al. 2001) and differences between population subgroups (Mossey and Shapiro 1982; Idler 1993; Joung, Stronks et al. 1995). It has been suggested that SRH is a very strong predictor of functional capacity (Idler and Kasl 1995; Ferraro, Farmer et al. 1997), future health problems (Kaplan, Goldberg et al. 1996; Möller, Kristensen et al. 1996) as well as mortality (Mossey and Shapiro 1982; Kaplan and Camacho 1983; Idler and Benyamini 1997; Mackenbach, Simon et al. 2002; Martikainen, Aromaa et al. 2002). Self-rated health has been suggested to have fair or good reliability (Martikainen, Aromaa et al. 1999).

*Psychological distress* (I) was measured using the General Health Questionnaire. GHQ is a self-report questionnaire for assessing current mental state, and it has been found to be a valid and reliable measure of mental health (Goldberg 1972; Goldberg, Gater et al. 1997; Martin 1999; Pevalin 2000). Originally the 60-item GHQ was used as a screening instrument (Goldberg 1972). However, shorter versions (GHQ12, GHQ28 and GHQ36) have been developed (Goldberg, Gater et al. 1997). The Health 2000 Survey used the GHQ 12-item scale to assess the participants’ experienced symptoms during the preceding weeks. The respondents were asked a series of 12 questions concerning psychological symptoms, such as a lack of concentration, sleeping difficulties, perceived stress and lack of self-confidence. The normal GHQ scoring method (0-0-1-1) for the four-item response scale (e.g. better than usual, as good as usual, worse than usual, much worse than usual). was used, giving a range of 0–12 for

the total score, which was accepted only if there were 10 or more valid items in the scale. In this thesis, the GHQ12 sum was dichotomised at the point 2/3, where a score of 3 or more was used to define those with psychological distress.

*Somatic morbidity* (I) was based on several questions inquiring whether a doctor had ever diagnosed the respondent as having a specified chronic somatic disease, and a complementary open-ended question coded on the basis of the ICD classification. This approach has been successfully used in many earlier Finnish national health surveys, and comparisons with simultaneous clinical examinations have shown (Heliövaara, Aromaa et al. 1993) that the agreement between open-ended self-reports and doctors' diagnoses depends on the condition and ranges from excellent (cardiovascular diseases) to moderate (musculoskeletal diseases). The respondents were considered to have a somatic disease if they reported at least one disease included in a list of 33 somatic disorders (Appendix A), ranging from serious congenital conditions to milder chronic somatic disorders. For some diseases additional criteria were applied. For example, asthma, arrhythmias, hypertension, back disorders, allergic and skin diseases as well as urinary infections were only considered to be present if the respondents reported being in a physician's care or using regular medication because of their disease. In those 902 persons considered to have at least one chronic somatic disease, the most common conditions were skin diseases (22%), serious allergies (15%), asthma (14%), back disorders (12%), other musculoskeletal disorders (12%), serious headache (10%), hypertension (6%) and diabetes (5%).

## 5.2.2 Health behaviour and BMI

In this thesis, self-reported smoking status was derived from four questions in the Health 2000 Survey: "Have you ever smoked?", "Have you ever smoked regularly (i.e. daily for at least one year)?", "When did you last smoke?" and "Have you smoked at least 100 times?" On the basis of this information five categories (II) were constructed that have also been used in other studies (Helakorpi, Prättälä et al. 2008). *Daily smokers* (II, IV and V) were defined as those who had smoked regularly for at least one year and most recently today or yesterday. The amount smoked was measured by asking the number of cigarettes smoked per day (II).

The measure of *heavy drinking* (III, IV and V) was based on information about both the frequency of drinking and the quantity consumed at a time for different types of alcohol during the past 12 months. The participants were asked to report average weekly consumption of "spirits", "wine" and "beer, cider and long-drinks" (in Finland "long drink" refers to a specific ready-mixed mild gin-flavoured alcoholic beverage). The amount of pure alcohol per week (g/week) was calculated and heavy drinkers were defined as women who consumed  $\geq 140$  g of pure alcohol and as men

who consumed  $\geq 280$  g of pure alcohol a week. The limits established for risk use of alcohol vary slightly between different countries, and the definition applied here follows the Finnish recommendations based on the occurrence of health problems at various levels of alcohol use, as published in the national evidence-based clinical practice guidelines for treatment of alcohol abuse (Salaspuro, Alho et al. 2005). It is also in line with a recent meta-analysis on the effects of alcohol intake on mortality (Di Castelnuovo, Costanzo et al. 2006).

*Body mass index BMI* (IV and V) was calculated from self-reported weight and height (weight/height<sup>2</sup>) and was categorised according to WHO recommendations (WHO 2000). The categories were: underweight  $< 18.5$  kg/m<sup>2</sup>, normal range 18.5–24.9 kg/m<sup>2</sup>, overweight 25–29.9 kg/m<sup>2</sup> and obesity 30+ kg/m<sup>2</sup>. In Substudy IV, BMI was used as three-class outcome variable: normal range, overweight and obesity (underweight respondents were removed from the analyses (7% in women, 1% in men). In the last Substudy (V), a two-class BMI was used: obese vs. others.

*Leisure-time physical activity* (IV and V) was based on the question “How often do you exercise in your leisure time so that you are at least slightly out of breath and sweating?” Three classes were constructed: “less than 1”, “1–3” and “4+” times a week. Health-wise, the recommendation for this kind of exercise is at least three times a week, lasting 20–60 minutes at time (Pate, Pratt et al. 1995), but our data did not allow for this categorization. Thus, some of those exercising 1–3 times a week follow the recommendation, but others do not.

*Use of vegetables* (IV and V) was based on the question “How often have you eaten vegetables and roots (not potatoes) during the past week (7 days) as such, grated or in fresh salads?” Three classes were constructed: “6–7”, “3–5” and “ $\leq 2$ ” times a week. Current recommendation is that vegetables should be eaten every day. In the European EFCOSUM project, the use of vegetables was put forward as one of the indicators of healthy nutrition (Steingrimsdottir, Ovesen et al. 2002).

### 5.2.3 Childhood circumstances

The measures of *mother's and father's educational level* (I–V) were based on the participant's response concerning his/her mother's and father's basic and vocational education. In most of the substudies, the parent with the higher level of educational attainment was chosen to indicate *parental education* (II–V). However, in the first Substudy, maternal and paternal educational levels were used as separate variables (I).

*Childhood family structure* (I–V) was based on the question, “When starting school (i.e. when you were about 7 years old), did you live...?” There were four response options: “at home with both your parents”, “with only one parent”, “with relatives

such as grandparents” and “in an orphanage or other institution”. The numbers were so small that participants reporting the last two options were combined into the category of “other living arrangement”. *Having siblings* (I) was based on the open-ended question, “How many siblings do you have/have you had (including stepsisters and stepbrothers, both dead and living)?” Two categories were constructed, i.e. “one or more” and “none”.

*Degree of urbanisation of childhood residence* (II–V) was based on the participants’ response concerning their place of residence during most of their childhood and categorised as “urban”, “semi-urban” or “rural” (Statistics Finland 2000). Those who had lived “abroad” for most of their childhood were categorised as a separate group.

Information on *childhood adversities* (I–V) was based on eleven items in a question which started as follows: “When you think about your growth years, i.e. before you were aged 16, did you experience...?” The items were: *long-term financial problems in the childhood family, regular parental unemployment, parental divorce, serious conflicts in the childhood family, maternal alcohol problems, paternal alcohol problems, maternal mental health problems, paternal mental health problems, own serious or chronic illness, parental serious illness or disability, own serious or chronic disease, and bullying at school*. In some substudies *parental mental health problems* were combined from mother’s and father’s mental health problems (III–V), and *parental alcohol problems* were constructed on the basis of questions concerning mother’s and father’s alcohol problems (IV and V). In the third Substudy (III), a three-class variable describing parental alcohol problems was used (classified as “none of the parents, “mother or father” and “both”).

*Parental smoking* (II) was based on the question, “Did your parent(s) smoke when you were between 13–15 years old?” The four response options were “no, neither of my parents smoked”, “yes, both parents smoked”, “only mother smoked” and “only father smoked”. Parents were considered here as those with whom the respondents lived at home (including stepfather or stepmother). If parental smoking status varied over time, the respondents were asked to answer according to the predominant situation.



**Table 2.** *The outcome variables, variables describing childhood and current circumstances, and other variables used in the original studies (I–V): definition, studies and literature reference for the measures.*

Variable	Definition	Study	Reference
<b>Outcome variables</b>			
Poor self-rated health (SRH)	Based on five-class self-assessed health ranging from good to poor. Average, poor or very poor self-rated health	I, V	
Psychological distress (GHQ)	Sum of the 12-item General Health Questionnaire points, Psychological distress = GHQ12 ≥ 3	I, V	Goldberg, 1972, Pevalin, 2000
Somatic morbidity	At least one of 33 listed somatic disorders, ranging from serious congenital conditions to milder chronic somatic disorders. For some diseases additional criteria were set (for more details, see study I).	I	ICD classification WHO
Daily smoking	Regular smoking for at least one year, most recently today or yesterday, and at least 100 times.	II, IV, V	Helakorpi et al; 2008
Heavy drinking	Pure alcohol ≥140 g/week (women), ≥280 g/week (men)	III, IV, V	Salaspuro et al; 2000 Di Castelnuovo, 2006
Overweight	BMI = 25–29.9 kg/m <sup>2</sup> , BMI = weight/height <sup>2</sup>	IV	WHO, 2000
Obesity	BMI = 30 + kg/m <sup>2</sup> , BMI = weight/height <sup>2</sup>	IV, V	WHO, 2000
<b>Childhood variables</b>			
Mother's education	Based on respondent's report on his/her mother's basic and vocational education	I	
Father's education	Based on respondent's report on his/her father's basic and vocational education	I	
Parental education	Highest educational level of parents based on previous variables (mother's and father's educational levels)	II–V	
Childhood family structure	With whom the respondent lived at age 7	I–V	
Degree of urbanisation of childhood residence	Degree of urbanisation level of the area in which the respondent lived most of his/her childhood	III–V	Statistics Finland, 2000
Childhood adversities			
Financial difficulties	Adversity prior to age of 16	I–V	<i>Living Conditions – Survey 1994</i>
Regular parental unemployment	Adversity prior to age of 16	I–V	
Parental divorce	Adversity prior to age of 16	I–V	
Serious conflicts	Adversity prior to age of 16	I–V	
Maternal serious alcohol problem	Adversity prior to age of 16	I–II	
Paternal serious alcohol problem	Adversity prior to age of 16	I–II	
Maternal mental health problem	Adversity prior to age of 16	I–II	
Paternal mental health problem	Adversity prior to age of 16	I–II	
Own serious or chronic illness	Adversity prior to age of 16	I–V	
Parental serious illness or disability	Adversity prior to age of 16	I–V	
Being bullied at school	Adversity prior to age of 16	I–V	
Parental smoking	Respondent's report on his/her parents' smoking habits when he/she was 13–15 years of age. Parents are considered here to be those persons with whom the respondent lived	II	
Parental alcohol problems (4 categories)	Constructed on the basis of respondent's reports of mother's and father's alcohol problems	III	

**Table 2.** *continues*

Variable	Definition	Study	Reference
Parental alcohol problems	Combined from mother's and father's alcohol problems reported by the respondent	IV, V	
Parental mental health problems	Combined from mothers' and father's mental health problems reported by the respondent	III–V	
Siblings	Number of siblings (including stepsisters and stepbrothers, both dead and living) reported by the respondent	I, II	
<b>Current life variables</b>			
Respondents' own educational level (4 categories)	Completed degree, for students expected level of education if higher than that already completed.	I–III	Aromaa et al, 2005
Respondents' own educational level (5 categories)	Completed degree, for students expected level of education if higher than that already completed.	IV	
Respondents' own educational level (3 categories)	Completed degree, for students expected level of education if higher than that already completed.	V	
Main economic activity	Taking up most of the time or accounting for most of income	II–V	
Current family structure (version 1)	Classified on the basis of marital status and household structure	III, IV	
Current family structure (version 2)	Combined on the basis of marital status, living arrangements and number of children	II	
Current family structure (version 3)	Classified on the basis of marital status and household structure (3 classes)	V	
Having children	Having children of one's own (men and women)	III–V	
Pregnant	Being pregnant at the time of the interview (women)	II	
Degree of urbanisation of current residence	Degree of urbanisation of residence at the time of data collection (1.7.2000)	III–V	Statistics Finland, 2000
Income quintiles	Self-reported household disposable income divided by the number of consumption units, derived by giving a value of 1.0 to the first adult in the household, 0.7 to any additional adult and 0.5 to each child.	III	OECD, 1982
<b>Other variables</b>			
Cigarette consumption	Average consumption of cigarettes per day	II	
Estimated age of smoking initiation	Derived by subtracting number of years of smoking from the respondent's age	II	
Use of vegetables	Frequency of eating vegetables/week (3 classes)	IV, V	Steingrimsdottir, Ovesen, Moreiras & Jacob, 2002
Leisure-time physical activity	Frequency of physical exercise in leisure time/week (3 classes)	IV, V	Pate, Pratt, Blair, Haskell, Macera, Bouchard et al., 1995
Use of sweets and sweet drinks	Frequency of consuming sweets and sweet drinks	IV	

## 5.2.4 Current circumstances

*Respondent's own education* was based on the highest completed level of education. Since many respondents were still studying (21% of the original participants;  $n=1,505$ ), the measure for students was based on the expected level of education, assuming the person would complete their ongoing studies and provided it was higher than the education they had already completed. Slightly different classifications were used in different substudies: Substudy I used a four-tiered classification between “only primary school”, “lower or upper secondary or lowest tertiary”, “lower degree level tertiary” and “higher degree level tertiary or higher”; studies II-IV a five-tiered classification between “only primary school”, “lower or upper secondary”, “lowest tertiary”, “lower degree level tertiary” and “higher degree level tertiary or higher”; and finally, Substudy V used a three-class variable: “primary”, “middle” and “high”. In the age group 30–39 (Substudy I), achieved educational level was determined using a four-class variable. In this thesis the person's educational level was chosen as the primary indicator of SEP, for it is often the first dimension of SEP that is established.

*Main economic activity* (II–V) was based on responses to the question on current main activity, defined as the one that took up most of the respondents' time or accounting for most of his/her income. The original eight response options were reduced to four categories “full-time or part-time employed”, “student”, “unemployed, retired or laid off”, and “other”. In men, the group “other” consisted mainly of those in military service (71%), and in women of those who looked after a family member or members at home (90%).

*Degree of urbanisation of current residence* was categorised as “big cities” (10 biggest cities by population in 2000), “other urban and semi-urban”, and “rural” municipalities (Statistics Finland 2000). *Current family structure* (II–V, three different versions) was determined by using variables describing marital status, household structure and number of children (II); alternatively, the presence of children was not taken into account (III–V). The measure was categorised as “married and child/children”, “married, no children”, “cohabiting and child/children”, “cohabiting, no children”, “living with parents”, “living with other(s) than a partner or parents” and “living alone” (II); “married”, “cohabiting”, “single” and living with other(s)” (III, IV); and “married or cohabiting”, “single” and “living with parents or others” (V). If the person belonged to more than one category, the highest one in the list was chosen. Women were categorised as *currently pregnant* based on self-report (II–IV) at the time of the interview, and both women and men were categorised as *having or not having children of their own* (III–V).

*Income quintiles* (III and V) were based on self-reported household disposable income divided by the number of consumption units, which was derived by giving a value of 1.0 to the first adult in the household, 0.7 to any additional adults and 0.5 to each child in the household (OECD 1982).

## 5.3 Statistical methods

### 5.3.1 General statistical methods

All analyses were performed using STATA statistical software versions 8.0 (StataCorp. 2003) and 9.1 (StataCorp. 2005). The data were weighted to take into account the sampling design and non-response in the Health 2000 Survey (Aromaa and Koskinen 2004) by using post-stratification weights (Lehtonen and Pahkinen 1994) constructed specifically for the dataset and by conducting the analyses using the survey procedures of the STATA software.

The analyses in studies I–IV are presented separately for men and women because there were statistically significant interactions between gender and some of the explanatory factors (reported in the individual studies). Interactions between age and all explanatory factors were also examined and reported if they were found to be statistically significant. In the last study (V) the analyses are presented together for men and women. In all five studies, subjects with missing information in the outcome measures were excluded from the data. In addition, some substudy-specific data restrictions were made (see Table 1).

The statistical methods used in the study include standard methods of population health research as well as multivariate statistical techniques (binomial and multinomial logistic regression analysis). For binomial logistic regression analysis (Substudies I, II, III and V) and multinomial regression analysis (Substudy IV), the results are presented in terms of odds ratios (ORs) and relative risk ratios (RRRs), respectively, together with 95% confidence intervals (95% CI). Potential explanatory and mediating variables were adjusted for first separately or in sets in a presumed causal order. Finally, all variables were simultaneously included in a logistic regression model.

### 5.3.2 Specific statistical methods in Substudies I–V

*In the first Substudy (I)*, the prevalence of different childhood circumstances and the distribution of poor self-rated health, psychological distress and somatic morbidity were calculated by gender and age group. In order to determine how different childhood adversities correlated with each other, pairwise Pearson correlations were calculated. Age-adjusted (direct standardisation) associations between the three

different health measures were also presented. In the second stage of the analysis, the associations between each health measure (poor SRH, psychological distress and somatic morbidity) and childhood circumstances were analysed with logistic regression analysis. The childhood determinants were added to the model sequentially, starting with the factors concerning early childhood (around age 7) and followed by variables describing circumstances at a later phase (prior to age 16). At the final stage, the models were adjusted for the respondent's own education.

*In the second Substudy (II)*, the distribution of smoking status, and in current daily smokers cigarette consumption and estimated age at onset of daily smoking, were calculated by gender, together with the prevalence of different childhood circumstances and current circumstances. The significance of the gender difference was calculated for all variables ( $\chi^2$ ). To assess the impact of the accumulation of childhood adversities on current daily smoking, the sum of the 11 childhood adversities was calculated, divided into five categories and their associations with the outcome measure tested. The associations between daily smoking and childhood as well as current determinants were analysed with logistic regression analysis. Items that did not show even a statistically indicative association ( $p > 0.1$ ) with daily smoking in either gender were removed from further analyses. However, parental mental health and alcohol problems were retained in the analyses due to very close statistically significant associations. Childhood determinants were incorporated in the model sequentially in order to assess pathways between them. Finally, the effect of the respondent's own education, current family structure and main activity were adjusted for.

*In the third Substudy (III)*, the associations between heavy drinking and potential explanatory factors were analysed using logistic regression analysis. At least marginally statistically significant determinants (Wald test  $p < 0.1$  for either or both genders, or if any category differed from the reference category at this significance level) were chosen for further modelling. Again, the childhood determinants were added to the model sequentially, starting with the factors concerning early childhood (around age 7), followed by variables describing the circumstances at a later phase (prior to age 16). At the next stage, the models were adjusted for the effect of the respondent's own education, main activity, current family structure and degree of urbanisation of current residence.

*In the fourth Substudy (IV)*, the associations between overweight/obesity and each potential explanatory factor (childhood circumstances, current circumstances and indicators of health behaviour) were analysed using multinomial logistic regression analysis, producing relative risk ratios (RRRs) for age-adjusted models. The first age-adjusted models were produced with all the data available ( $n = 1,369$ ). At least marginally statistically significant determinants ( $p < 0.1$  for either or both genders, or if any of the categories differed from the reference category at this significance

level) were chosen for further modelling, where only subjects who answered both the interview and the questionnaire (n=1,158) were included: the model based on this smaller population allowed for a proper assessment of the changes between the models. The childhood determinants were added to the model sequentially, starting with the factors concerning early childhood (around age 7), followed by variables describing the circumstances at a later phase (prior to age 16). At the next stage, the models were adjusted for the effect of adult characteristics and in addition, for statistically significant behavioural indicators (physical activity, use of vegetables).

*In the fifth Substudy (V)*, the distribution of SRH by level of education in young adults was first calculated. Secondly, the associations between all potential explanatory factors and poor SRH and education were calculated and the p-values (Chi2 and Wald test) were presented to illustrate the significance of the associations. Explanatory factors associated with *both* poor SRH and the respondent's own education at the significance level  $p < 0.25$  were chosen for further modelling.

Next, the results on the contribution of explanatory factors to educational differences in poor SRH were calculated. In the first model, the educational differences in poor SRH were adjusted for age and gender. Additional explanatory factors were added (first one at a time and then in sets) to obtain further models. The observed reduction in the strength of association between education and SRH from the first model to the subsequent models represents the contribution of the explanatory factor(s) to educational differences in poor SRH. The percentage reduction was calculated as in previous studies (Stronks, van de Mheen et al. 1996; Laaksonen, Roos et al. 2005; van Oort, van Lenthe et al. 2005; Sainio, Martelin et al. 2007):

$$(\text{OR}_{(\text{base model})} - (\text{OR}_{(\text{base model} + \text{intermediate factor(s)})}) / (\text{OR}_{(\text{base model})} - 1) \times 100\%.$$

The final step was to calculate to what extent childhood circumstances, current circumstances and health behaviour had shared effects on educational health differences. The shared effects of two sets of explanatory variables were calculated by first summing up the reductions in the strength of association between education and SRH observed when including the two sets of variables separately in the age-adjusted model. The reduction observed when including both sets of explanatory factors simultaneously in the age-adjusted model was subtracted from this sum. The result of this subtraction represents the shared effect of the two sets of variables. The extent to which the effect of childhood circumstances was shared with current circumstances and/or health behaviour was estimated as the ratio between the shared effect and the effect of childhood circumstances alone.

## **5.4 Ethical considerations**

Ethical and data protection issues received full and appropriate consideration during all phases of this research. Generally accepted ethical guidelines have been followed in the use and reporting of the data. The instructions issued by the National Advisory Board on Research Ethics and data protection authorities have been followed at all phases and with all data sources.

Data protection and the appropriate handling and storage of all the data and materials collected has been a major consideration at all stages of the Health 2000 Survey. Every possible precaution has been taken to prevent unauthorised access. High standards of data security have been maintained throughout. All personal identification codes have been deleted in the data so that individual participants cannot be identified by the researchers in the datasets released to them for analysis. However, since personal identification numbers are needed for follow-up purposes as well as for linking with other data, they are accessible to a small number of authorised personnel for these specified purposes.

The plans and protocols for the Health 2000 Survey have been submitted for approval to the relevant ethical committees. The application was reviewed by the National Public Health Institute's Ethical Committee in September 1999. Following changes in legislation, a more detailed project plan was submitted to the Ethical Committee for Research in Epidemiology and Public Health at the Hospital District of Helsinki and Uusimaa (HUS) in May 2000. At both these stages, the plans received favourable opinions.

All the necessary permissions and informed consent of the subjects have been acquired when the data collection was conducted (Heistaro 2005). An information letter was handed out to the subjects in the home interviews conducted by staff of Statistics Finland and later in the health examination. In both of these situations trained staff were available to answer any questions. Further, the subjects were asked to sign informed consent forms.

## 6 RESULTS

### 6.1 Characteristics of the study population

#### 6.1.1 Childhood and current circumstances

The distributions for the most frequently used variables describing childhood and current circumstances are presented for young adults aged 18–29 years in Table 3. In the first Substudy a broader age range was used (18–39 years), and the distributions of the measures used for the older age-group (30–39 years) are presented in Substudy I, Table 1.

Paternal and maternal educational levels were quite evenly distributed between the four classes (Table 3; Substudy I, Table 1). The numbers in the lowest educational category were slightly higher than in the other categories, particularly in the case of fathers. When information on paternal and maternal education was combined, the three categories were almost equally common and primary level education was the least common category. The most common family structure was having lived with two parents in childhood, and most of the participants had had siblings as well (in both cases over 90% of the participants).

Commonly reported childhood adversities included paternal alcohol problem, long-term financial problems in the family, parental divorce, serious conflicts within the family, parental serious illness or disability and bullying at school (13–29%) (Table 3; Substudy I, Table 1). Less frequently (3–8%) reported problems included parental mental health problems, maternal alcohol problem and respondent's own serious or chronic illness in childhood. In general, women reported childhood adversities more often than men. A statistically significant gender difference was seen in responses concerning long-term financial problems, parental alcohol problems and serious conflicts within the family ( $p < 0.05$ ).

In the age group 18–29 years, the pairwise correlations between different childhood adversities were  $r \leq 0.4$ . The strongest correlation coefficients were found between serious conflicts within the family and paternal alcohol problem ( $r = 0.40$ ), between parental divorce and serious conflicts within the family ( $r = 0.33$ ), and between long-term financial problems and regular parental unemployment ( $r = 0.33$ ). No negative correlations were found between childhood adversities. Over half of the respondents had lived most of their childhood in urban or semi-urban municipalities (Table 3).



**Table 3. Distribution of childhood circumstances and current circumstances by gender in young adults aged 18–29 years (%).**

Childhood circumstances	Men	Women	Current circumstances	Men	Women
<b>Paternal education</b>			<b>Own education</b>		
Secondary school graduate	15	16	Higher-degree level tertiary or higher	15	21
Middle level education	18	18	Lower-degree tertiary	24	34
Primary level and some vocational education	24	25	Secondary	51	37
Primary level education only	31	29	Primary	10	7
Didn't live with father	7	9	Information missing	0.2	0.4
Don't know or information missing	5	4	p <sup>1</sup>		0.000
p <sup>1</sup>		0.848			
<b>Maternal education</b>			<b>Main activity</b>		
Secondary school graduate	21	22	Full-time or part time employed	68	53
Middle level education	21	24	Student	17	25
Primary level and some vocational education	23	24	Unemployed or laid off	9	8
Primary level education only	29	25	Other	6	13
Didn't live with mother	1	1	Information missing	0.0	0.4
Don't know or information missing	6	4	p <sup>1</sup>		0.000
p <sup>1</sup>		0.368			
<b>Highest parental education</b>			<b>Income quintiles</b>		
Secondary school graduate	25	26	1 lowest	17	21
Middle level education	24	25	2	19	21
Primary level and some vocational education	28	29	3	21	19
Primary level education only	19	17	4	19	22
Didn't live with parents	4	3	5 highest	24	18
Don't know or information missing	0.4	0.5	Information missing	0.0	0.0
p <sup>1</sup>		0.796	p <sup>1</sup>		0.000
<b>Family structure</b>			<b>Family structure</b>		
Two parents	92	90	Married	14	20
One parent	8	9	Cohabitation	32	39
Other	0.4	0.5	Living alone	27	25
Information missing	0.0	0.4	Living with own parents	26	11
p <sup>1</sup>		0.380	Living with other(s)	1	5
			Information missing	0.1	0.5
			p <sup>1</sup>		0.000
<b>Number of siblings</b>			<b>Children</b>		
None	7	6	Yes	17	26
One	43	38	No	83	74
Two or more	50	56	Information missing	0.0	0.4
Information missing	0.0	0.7	p <sup>1</sup>		0.000
p <sup>1</sup>		0.015			
<b>Adversities<sup>2,3</sup></b>			<b>Degree of urbanisation of place of residence</b>		
Long-term financial problems in the family	15	19	Big cities	40	47
Regular parental unemployment	10	12	Small cities and semi-urban municipalities	41	38
Parental divorce	19	21	Rural municipalities	18	14
Paternal alcohol problem	14	20	Information missing	0.0	0.0
Maternal alcohol problem	3	8	p <sup>1</sup>		0.257
Paternal mental health problem	3	5			
Maternal mental health problem	3	6			
Parental serious illness or disability	13	15			
Serious conflicts within the family	19	29			
Own serious or chronic long-term illness	3	4			
Bullying at school	22	27			
<b>Urbanisation level of the place of residence</b>					
Urban	52	58			
Semi-urban	17	17			
Rural	28	23			
Abroad	2	1			
Information missing	0.1	0.5			
p <sup>1</sup>		0.014			
			Sample (N)	981	913
			Participants (N)	765	740
			Participation rate (%)	78	81

<sup>1</sup> Significance of the difference between genders (Chi2).

<sup>2</sup> Childhood adversities were asked in the questionnaire (n=1,282; 680 women, 602 men)

<sup>3</sup> The significance of the gender difference concerning childhood adversities are reported in Substudy I.

The distributions for young adults' current circumstances are presented in Table 3. Just over 50% of men and 37% of women had a secondary degree, which was the most common educational category. Just under 10% of the respondents had no education beyond primary level education. Almost ten per cent of the young adults were still studying, and a slightly larger proportion of men (68%) than women (53%) were currently full-time or part-time employed. Almost one-tenth of the respondents reported being unemployed. Cohabiting was the most common family structure (32% in men, 39% in women), although relatively large numbers were married and one-quarter lived alone. Among men 26% and among women 11% lived with their parents (Table 3). Approximately one-quarter of the women had children of their own, in men the proportion was smaller (17%). Over 80% of the respondents lived in urban or semi-urban municipalities.

### 6.1.2 Health in the young adult population

The prevalence of poor SRH, psychological distress and somatic morbidity in young adults as well as their associations were examined in Substudy I. Each of these three health indicators was statistically significantly more common in the older (30–39 years of age) than in the younger age group (18–29 years of age), as can be seen in Table 4 (see also Substudy I, Table 2). Poor SRH was more common in men (14%) than in women (12%) ( $p < 0.05$ ), whereas psychological distress and somatic morbidity were more common in women than in men ( $p < 0.001$ ): 16% of men and 22% of women were classified as having psychological distress, and 26% of men and 33% of women had at least one of the somatic disorders examined in this study.

**Table 4.** *Prevalence of poor SRH, psychological distress and somatic morbidity by gender and age group and health status according to the other health indicators (%).*

Measure of health	Proportion (%) of respondents having the health problem			Proportion <sup>1</sup> (%) of those having the health problem who also had		
	18–29	30–39	18–39 <sup>1</sup>	Poor SRH	Psychological distress	Somatic morbidity
<b>Men:</b>						
Poor SRH	11	17	14	-	40	49
Psychological distress	13	18	16	35	-	35
Somatic morbidity	22	31	26	26	22	-
At least one of the health problems	39	49	44			
<b>Women:</b>						
Poor SRH	9	14	12	-	52	52
Psychological distress	20	25	22	27	-	42
Somatic morbidity	32	36	34	18	27	-
At least one of the health problems	48	55	52			

<sup>1</sup> age group 18–39, age-adjusted

Having any of these three health problems was associated with an increased probability of having another health problem as well (Table 4; Substudy I, Table 2). For example, those with poor SRH were more likely to have psychological distress (40% in men, 52% in women) than the whole sample (16% and 22%, respectively). However, the correlation coefficients between the three measures of health were quite low: Pearson  $r=0.09$  between psychological distress and somatic morbidity,  $r=0.16$  between poor SRH and somatic morbidity, and  $r=0.25$  between poor SRH and psychological distress.

### 6.1.3 Health behaviour and BMI in the young adult population

Substudies II–IV focused on analysing the prevalence of three indicators of health behaviour in young adults. The distributions of smoking and drinking status, heavy drinking as well as classified BMI are presented in Table 5. The pairwise correlations between the three measures of health behaviour were rather low, all  $r<0.16$ .

Daily smoking was significantly more common in men than in women ( $p<0.001$ ): 36% of men and 24% of women smoked daily, but there was no clear gender difference in occasional smoking (Table 5; Substudy II, Table 1). A higher proportion of women than men were non-smokers ( $p<0.001$ ). Men smoked substantially more than women: in daily smokers almost one-third of men, but only one-tenth of women smoked more than 20 cigarettes a day, whereas lighter smoking (1–9 cigarettes per day) was more common in women. The majority of women and men (ca. 80%) had started daily smoking before the age of 19. However, in our data there were 80 persons who had not yet reached this age and who had never smoked (Substudy II, Table 1).

Almost 90% of young adults aged 18–29 years reported that they used alcohol (Table 5; Substudy III, Table 1). Most abstainers were life-long non-drinkers and only a small proportion (2% in both women and men) were ex-drinkers. Heavy drinking based on the specified limits of 280g of pure alcohol/week in men and 140g/week in women was more common in men (8%) than in women (5%) ( $p=0.035$ ).

Almost three-quarters of women and two-thirds of men were normal weight ( $18.5\leq\text{BMI}<25$ ) (Table 5; Substudy IV, Table 2). Being underweight was more common in women (7%) than in men (1%) ( $p<0.001$ ), and the prevalence of overweight ( $25\leq\text{BMI}<30$ ) was higher in men than in women: 28% and 14%, respectively ( $p<0.001$ ). There was no gender difference in obesity ( $\text{BMI}\geq 30$ ) as 8% of men and 6% of women were obese ( $p=0.402$ ). Extreme obesity ( $\text{BMI}\geq 35$ ) was rare in both men (1%) and women (2%).

**Table 5.** *Distribution<sup>1</sup> (%) of current smoking status, current drinking status and heavy drinking<sup>3</sup> as well as classified BMI (WHO) in women and men aged 18–29 years. See varying sample sizes due to data restrictions in Table 1.*

Variable	Men	Women
<b>Current smoking status</b>		
Current daily smokers	36	24
Occasional smokers	9	10
Ex-daily smokers given up 1–12 months ago	4	5
Ex-daily smokers given up > year ago	6	7
Non-smokers	46	54
Smoking status missing	0.1	0.5
p <sup>2</sup>		0.000
n = 1,505	765	740
<b>Current drinking status</b>		
Life-long non-drinker	8	10
Ex-drinker	2	2
Current drinker	88	86
Drinking status missing	1	2
p <sup>2</sup>		0.469
n = 1,234	602	632
<b>Heavy drinking<sup>3</sup></b>		
No	92	85
Yes	8	5
Information missing	0.3	0.3
p <sup>2</sup>		0.082 <sup>4</sup>
n = 1,234	632	602
<b>BMI</b>		
Underweight BMI < 18.5	1	7
Normal range 18.5 ≤ BMI ≤ 24.9	63	73
Overweight 25 ≤ BMI ≤ 29.9	28	14
Obesity BMI ≥ 30	8	6
p <sup>2</sup>		0.000
n = 1,426	762	664

<sup>1</sup> Prevalence is weighted to represent the whole population.

<sup>2</sup> Significance of the gender difference.

<sup>3</sup> For men ≥ 280g of pure alcohol/week and for women ≥ 140g of pure alcohol/week.

<sup>4</sup> Not including the group with missing information, p=0.035.

## **6.2 Determinants of poor SRH, somatic morbidity and psychological distress (I)**

This chapter presents the main results of Substudy I, beginning with the age-adjusted associations between childhood circumstances and the health indicators (6.2.1) and proceeding then to the effect of the respondent's own education on these associations (6.2.2). The results combine the most important age-adjusted associations and education-based mediating effects for young adults aged 18–39 years. Only statistically significant associations are shown in Figures 4–6; other results can be found in Substudy I, Tables 3–5.

### **6.2.1 Age-adjusted associations between childhood circumstances and health indicators**

No consistent pattern was observed for the effect of parental education on the three health indicators, but it varied depending on the health measure. In both genders, maternal education seemed to be associated with poor SRH, whereas paternal education did not show a statistically significant association with poor SRH (Table 6; Substudy I, Tables 3 and 4). On the other hand, maternal education was not associated with psychological distress, but having lived with a highly educated father appeared to increase the risk of psychological distress. In addition, in men low paternal education, and in women low maternal education, increased the risk of somatic morbidity (OR=1.63 in both cases).

Childhood family structure was an important determinant of poor SRH and psychological distress, but only in men (Table 6; Substudy I, Tables 3 and 4). Living with one parent in childhood clearly increased the risk of both poor SRH (OR=2.15) and psychological distress (OR=2.15) as compared to living with both parents. In women, no statistically significant associations were seen between health indicators and childhood family structure, and somatic morbidity was not associated with childhood family structure in either men or women (Table 6; Substudy I, Table 5). The number of siblings did not predict psychological distress or poor SRH, but somatic morbidity was slightly more common among men with two or more siblings as compared to those with only one sibling.

Regarding specific childhood adversities experienced prior to 16 years of age, the respondent's own serious or chronic illness was associated with all three health indicators in both genders (Table 6; Substudy I, Tables 3–5). Long-term financial problems, serious conflicts within the family and having been bullied at school were statistically significantly associated with poor SRH and psychological distress in both men and women. Having been bullied at school was also associated with a higher risk of somatic morbidity in men (OR=1.46). Parental serious illness or disability

predicted poor SRH in men and women (OR = 1.90 and OR = 1.71, respectively), and in men it also predicted psychological distress (OR = 2.29).

Parental alcohol problems predicted psychological distress in both genders and poor SRH in women. In addition, maternal alcohol problems were associated with somatic morbidity in women (OR = 1.89). Paternal mental health problems seemed to be a statistically significant predictor of health as it was associated with psychological distress in both genders (OR = 2.89 in men and OR = 2.63 in women), and in men also with poor SRH (OR = 3.31). Maternal mental health problems were statistically significant for women; both poor SRH (OR = 3.10) and psychological distress (OR = 2.57) were associated with maternal mental health problem. Parental divorce predicted poor SRH in women (OR = 1.66) and psychological distress in men (OR = 1.54), and parental unemployment predicted somatic morbidity in both men and women (OR = 1.62 and OR = 1.70, respectively).

**Table 6.** *Age-adjusted odds ratios (OR) for poor SRH, psychological distress and somatic morbidity by childhood circumstances in men and women.*

Variable	Poor SRH		Psychological distress		Somatic morbidity	
	Men	Women	Men	Women	Men	Women
<b>Paternal education PE</b>						
Secondary school graduate	1.00	1.00	1.00	1.00	1.00	1.00
Middle level education	1.10	0.99	0.38*	0.51*	1.19	1.29
Primary level and some vocational	1.23	1.28	0.60*	0.52*	1.63*	1.23
Primary level education only	1.21	1.21	0.47*	0.57*	1.41	1.00
<b>Maternal education ME</b>						
Secondary school graduate	1.00	1.00	1.00	1.00	1.00	1.00
Middle level education	1.78*	2.10*	0.86	0.79	1.20	1.43
Primary level and some vocational	1.14	2.27*	0.77	0.70	1.09	1.04
Primary level education only	1.16	2.15*	0.81	0.66	1.38	1.63*
<b>Childhood family structure ChFS</b>						
Two parents	1.00	1.00	1.00	1.00	1.00	1.00
Single-parent family	2.15*	1.09	2.15*	1.46	1.13	1.03
Other	1.14	3.07	0.48	1.45	2.70	2.53
<b>Number of siblings SI</b>						
Two or more	1.00	1.00	1.00	1.00	1.00	1.00
One	0.97	0.85	1.20	0.90	0.73*	1.00
None	0.67	1.46	0.82	1.14	0.84	0.98
<b>Childhood adversities<sup>1</sup> CA</b>						
Own serious or chronic illness	3.76*	4.32*	2.49*	1.89*	6.49*	2.99*
Bullying at school	1.72*	2.43*	3.29*	1.50*	1.46*	1.29
Long-term financial problems	2.06*	1.95*	3.30*	1.74*	1.19	1.31
Serious conflicts within the family	1.48*	2.28*	2.36*	1.75*	1.30	1.18
Parental serious illness or disability	1.90*	1.71*	2.29*	1.22	1.05	1.29
Paternal alcohol problems	1.34	1.46*	1.79*	1.45*	1.30	1.08
Maternal alcohol problems	1.04	2.40*	3.84*	1.83*	0.86	1.89*
Paternal mental health problems	3.31*	1.82	2.89*	2.63*	2.00	0.93
Maternal mental health problems	0.86	3.10*	1.69	2.57*	1.41	1.40
Parental divorce	1.45	1.66*	1.54*	1.23	0.94	0.98
Regular parental unemployment	1.24	1.11	1.72	1.24	1.62*	1.70*

\* p < 0.05

<sup>1</sup> Reference group for each CA is those respondents who answered "no" or "don't know" to the particular question.

### 6.2.2 The effect of education on the associations

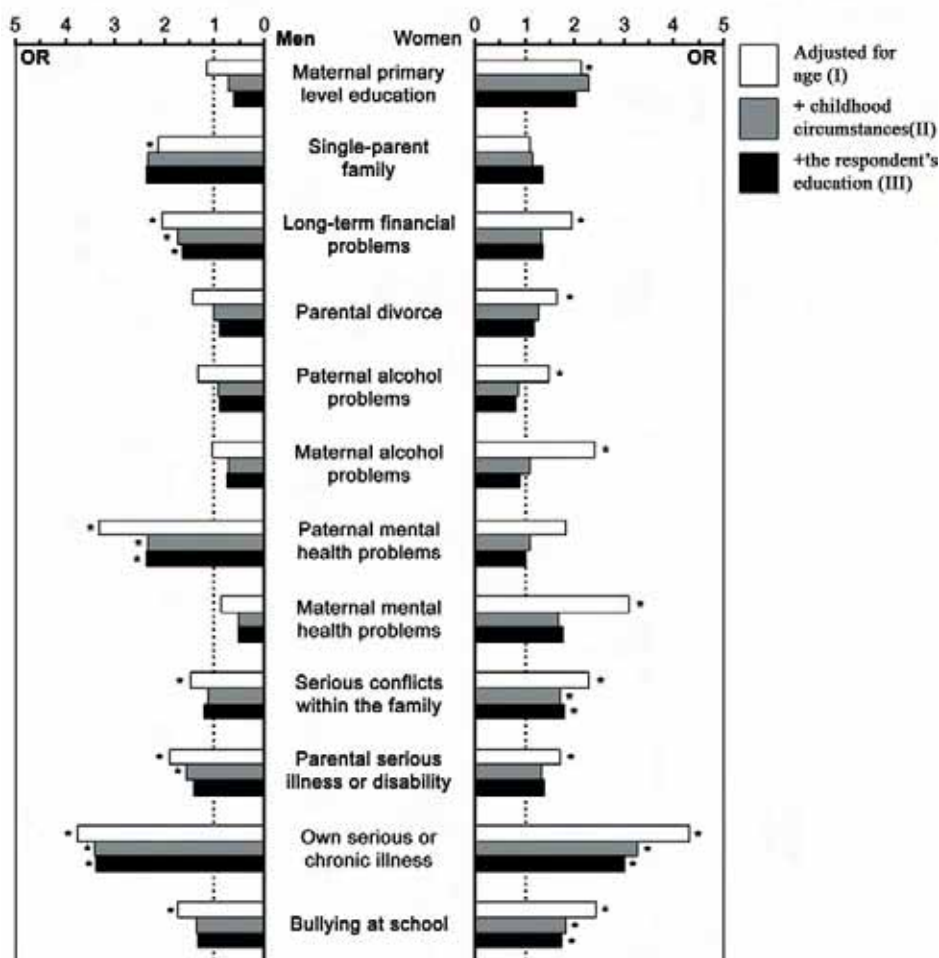
Most of the associations between poor SRH and childhood circumstances were attenuated after adjusting for all childhood circumstances (Figure 3, Model II; Substudy I, Table 3). However, the respondent's own chronic or long-term illness remained strongly associated with poor SRH. In addition, serious conflicts within the family and bullying at school remained statistically significantly related to poor SRH in women and long-term financial problems, paternal mental health problems and parental serious illness or disability in men. The associations between childhood circumstances and poor SRH remained relatively unchanged after controlling for the respondent's own education (Figure 3, Model III).

Almost all childhood adversities were found to be statistically significantly associated with psychological distress in the age-adjusted models for both genders (Figure 4, Model I; Substudy I, Table 4). These associations attenuated when all childhood circumstances were included in the model, but many of them remained statistically significant (Figure 4, Model II). In men, long-term financial problems, maternal alcohol problems, bullying at school and parental serious illness or disability, and in women parental mental health problems were significantly associated with psychological distress after these adjustments. Adjusting for the respondent's own education had only a minor effect on the associations between childhood circumstances and psychological distress (Figure 4, Model III).

Only a few childhood adversities were statistically significantly associated with somatic morbidity in the age-adjusted model (Figure 5, Model I; Substudy I, Table 5), and adding all childhood conditions to the model at the same time did not considerably change the results. However, the associations between regular parental unemployment and somatic morbidity, and in women between maternal alcohol problems and somatic morbidity, were no longer statistically significant (Figure 5, Model II). The associations remained relatively unchanged after controlling for the respondent's own education (Figure 5, Model III). The respondent's own chronic or long-term illness in childhood predicted somatic morbidity in early adulthood despite all adjustments, as did having been bullied at school in men. In women, low maternal education predicted somatic morbidity despite all the adjustments (Figure 5, Model III).



**Figure 3. Poor self-rated health (SRH) by childhood circumstances<sup>1</sup> in men and women aged 18–39 years (OR). Adjusted for age, childhood circumstances<sup>2</sup> (maternal education, paternal education, childhood family structure, number of siblings and childhood adversities) and the respondent's own education.**

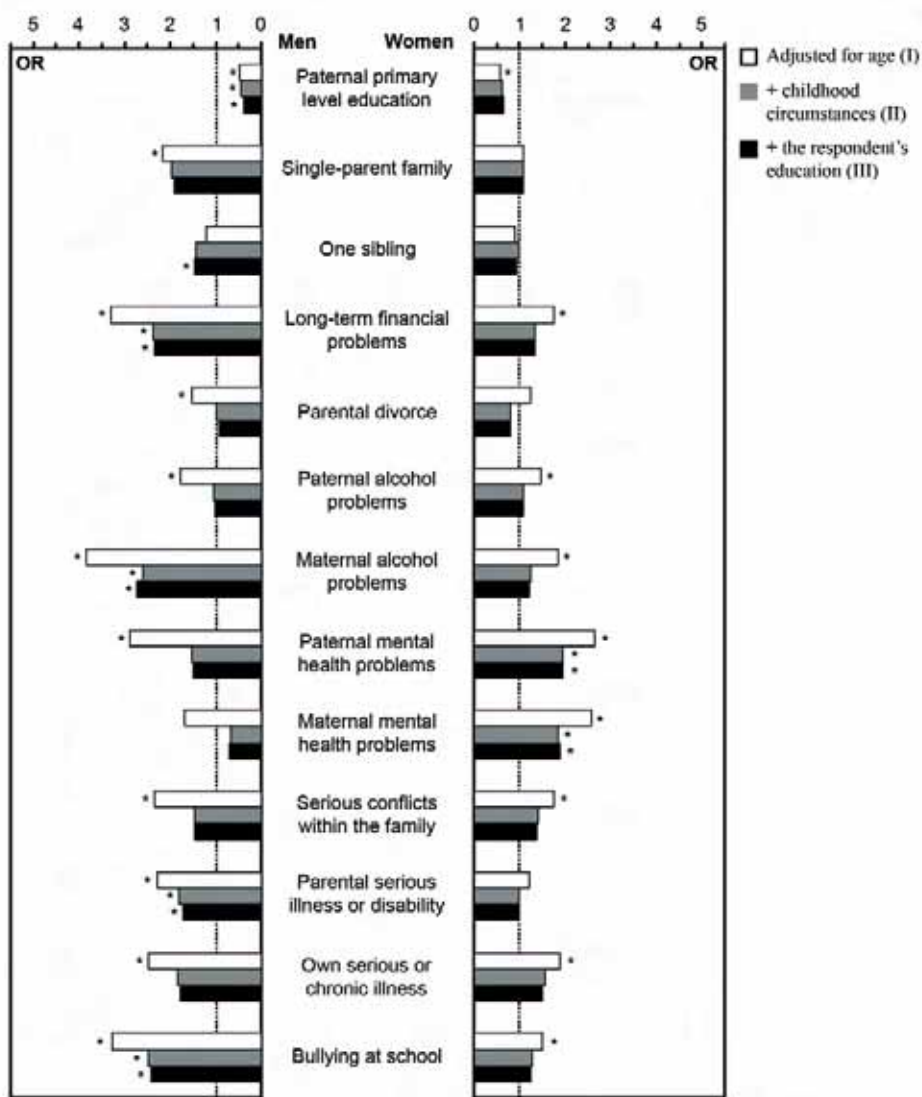


\*  $p < 0.05$

<sup>1</sup> Only childhood circumstances statistically significant for either gender are presented (for others, see Substudy I, Table 3).

<sup>2</sup> Maternal primary level education: compared to the highest educational category (secondary degree); single-parent family: compared to two-parent family; adversities compared to those who did not report the specific problem in childhood.

**Figure 4.** *Psychological distress (GHQ $\geq$ 3) by childhood circumstances<sup>1</sup> in men and women aged 18–39 years (OR). Adjusted for age, childhood circumstances<sup>2</sup> (maternal education, paternal education, childhood family structure, number of siblings and childhood adversities) and the respondent's own education.*

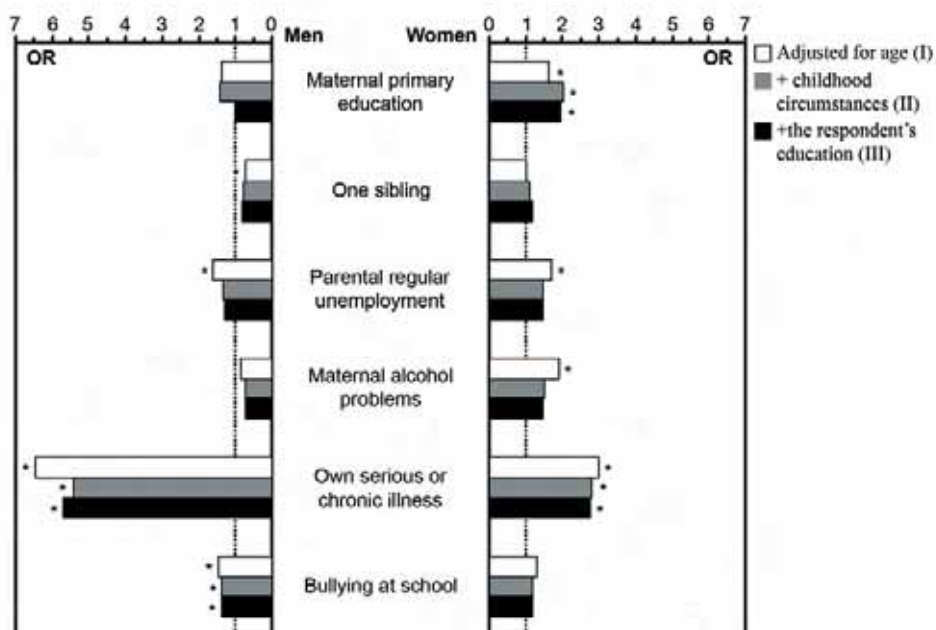


\*  $p < 0.05$

<sup>1</sup> Only childhood circumstances statistically significant for either gender are presented (for others, see Substudy I, Table 4).

<sup>2</sup> Paternal primary level education compared to the highest educational category (secondary degree); single-parent family compared to two-parent family; one sibling compared to those with none. Adversities compared to those who did not report the specific problem in childhood.

**Figure 5. *Somatic morbidity by childhood circumstances<sup>1</sup> in men and women aged 18–39 years (OR). Adjusted for age, childhood circumstances<sup>2</sup> (maternal education, paternal education, childhood family structure, number of siblings and childhood adversities) and the respondent's own education.***



\*  $p < 0.05$

<sup>1</sup> Only childhood circumstances statistically significant for either gender are presented (for others, see Substudy I, Table 5).

<sup>2</sup> Maternal primary level education compared to the highest educational category (secondary degree); one sibling compared to those with none. Adversities compared to those who did not report the specific problem in childhood.

### 6.3 Determinants of smoking, heavy drinking and obesity (II–IV)

This chapter presents the main results of Substudies II–IV: first, the age-adjusted associations between childhood circumstances and indicators of health behaviour (6.3.1) and secondly, the effect of the respondent's own education and other current circumstances on these associations (6.3.2). Only statistically significant associations are presented in Figures 6–8 ( $p < 0.05$ ); other results can be found in Substudies (II–IV).

### 6.3.1 Age-adjusted associations between childhood circumstances and indicators of health behaviour

Parental education was an important predictor of smoking and obesity in early adulthood, whereas its association with heavy drinking seemed to be weaker. Parental education had a strong negative association with daily smoking in both genders after adjustment for age: the lower the level of parental education, the higher the prevalence of daily smoking in both genders (Table 7; Substudy II, Tables 4 and 5). A statistically significant but rather inconsistent association was observed between parental education and obesity in both genders. Parental education had no clear association with overweight in men, but in women a negative association was seen: the lower the level of parental education, the higher the prevalence of overweight (Table 8; Substudy IV, Table 3). Heavy drinking did not show a clear association with parental education as only parental middle level education increased the risk of heavy drinking at the  $p < 0.1$  significance level (OR=1.93). Living in a single-parent family in childhood predicted daily smoking (although only in the younger age group 18–24 years) and obesity in women and heavy drinking in men in the age-adjusted models (Table 7; Substudy II, Tables 4 and 5; Substudy III, Table 2; Substudy IV, Table 3).

Daily smoking was predicted by a greater number of childhood adversities in women than in men (Table 7). In women long-term financial problems, serious conflicts within the family, maternal alcohol and mental health problems as well as parental divorce predicted daily smoking in the age-adjusted models. In men, only parental divorce and regular parental unemployment were statistically significantly associated with daily smoking in early adulthood (Table 7).

Daily smoking also varied strongly according to parental smoking in both genders, as the respondents whose both parents smoked were the most likely to be daily smokers themselves (OR=3.27 in men and OR=3.74 in women). Furthermore, those men whose father (OR=1.67) or mother (OR=1.97) smoked, and those women whose mother (OR=2.71) smoked were more likely to be daily smokers than those whose parents did not smoke at all (Table 7). The more childhood adversities the respondents reported, the more likely they were to smoke daily. The relationship between daily smoking and the number of reported childhood adversities was particularly strong in women ( $p=0.000$ ) (See Substudy II, Table 2 for these cumulative results).

Heavy drinking was also associated with childhood adversities. In women, long-term financial problems (OR=2.43) and serious conflicts within the childhood family (OR=2.31) were associated with heavy drinking and in men, parental divorce (OR=2.16), parental mental health problem (OR=2.85), parental serious illness or disability (OR=3.31) and having been bullied at school (OR=2.00) predicted heavy drinking (Table 7). There were statistically significant associations between parental

alcohol problems and heavy drinking in both genders in the age-adjusted models: those men and women whose both parents had alcohol problems were at the highest risk of being heavy drinkers themselves (OR=4.67 and OR=4.97, respectively).

Childhood adversities seemed to be important predictors of overweight and obesity only in women (Table 7). In women, regular parental unemployment (OR=2.09) was statistically significantly associated with overweight, whereas parental alcohol problems (OR=2.71) and mental health problems (OR=2.28) and being bullied at school (OR=3.13) were associated with obesity. In men, none of the childhood adversities were statistically significantly associated with overweight or obesity.

**Table 7. Age-adjusted odds ratios (OR) for indicators of health behaviour by childhood circumstances in men and women aged 18–29 years.**

Variable	Daily smoking		Heavy drinking <sup>d</sup>		Obesity <sup>e</sup>	
	Men	Women	Men	Women	Men	Women
<b>Parental education<sup>1</sup> PE</b>						
Secondary school graduate	1.00	1.00	1.00	1.00	1.00	1.00
Middle level education	1.58*	2.01*	1.93	1.27	3.37*	0.94
Primary level and some vocational	1.65*	2.26*	1.60	1.13	2.72	2.55*
Primary level education only	2.06*	2.13*	1.22	0.21	2.85*	4.20*
<b>Childhood family structure ChFS</b>						
Two parents	1.00	1.00	1.00	1.00	1.00	1.00
Single-parent family	1.41	2.19* <sup>3</sup>	3.23*	1.21	0.85	2.40*
<b>Childhood adversities<sup>2</sup> CA</b>						
Own serious or chronic illness	-	-	0.83	1.71	-	-
Bullying at school	-	-	2.00*	1.16	1.35	3.13*
Long-term financial problems	1.14	1.78*	1.40	2.43*	1.02	1.83
Serious conflicts within the family	1.27	1.74*	1.01	2.31*	1.52	1.86
Parental serious illness or disability	-	-	3.31*	0.91	-	-
Paternal alcohol problems	1.49	1.50	-	-	-	-
Maternal alcohol problems	1.70	2.57*	-	-	-	-
<i>Parental alcohol problem</i>	-	-	-	-	1.68	2.71*
Paternal mental health problems	0.78	1.61	-	-	-	-
Maternal mental health problems	0.87	2.17*	-	-	-	-
<i>Parental mental health problems</i>	-	-	2.85*	1.71	2.17	2.28*
Parental divorce	1.79*	2.90*	2.16*	1.17	1.57	1.19
Regular parental unemployment	1.79*	1.66	1.05	2.00	0.73	1.24
<b>Parental smoking PS</b>						
Neither parents	1.00	1.00	-	-	-	-
Both parents	3.27*	3.74*	-	-	-	-
Father smoked	1.67*	1.42	-	-	-	-
Mother smoked	1.97*	2.71*	-	-	-	-
<b>Parental alcohol problems PAB</b>						
Neither parents	-	-	1.00	1.00	-	-
Mother or father	-	-	1.17	0.79	-	-
Both	-	-	4.67*	4.97*	-	-

\* p<0.05, – OR not calculated

<sup>1</sup> Highest educational level of parents,

<sup>2</sup> Reference group for each CA is those respondents who answered "no" or "don't know" to the particular question.

<sup>3</sup> Interaction AGE\*ChFS, p=0.013. Respondents from single-parent families differed significantly from those with two parents only in the younger age group (18–24 years of age) (for the younger OR=2.72\* and for the older one OR=1.64).

<sup>4</sup> For men ≥280g of pure alcohol/week and for women ≥140g of pure alcohol/week.

<sup>5</sup> BMI≥30, normal weight persons (BMI=18.5–24.9) is the reference category.

### 6.3.2 The role of current circumstances in the associations between childhood circumstances and indicators of health behaviour

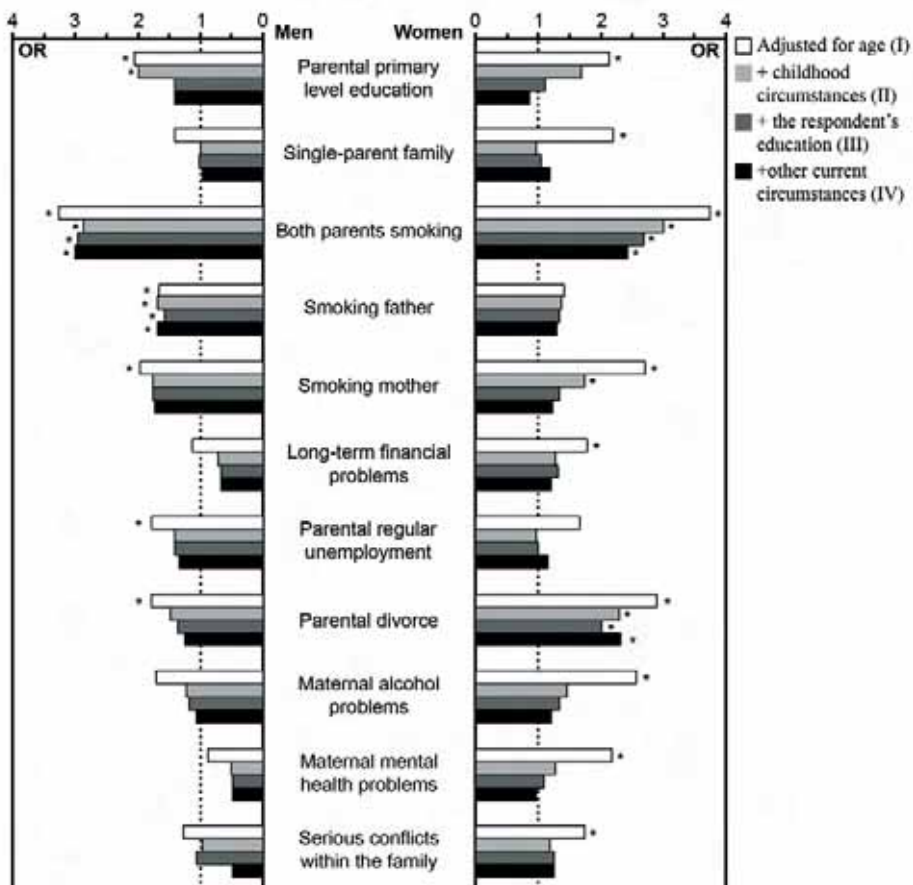
#### Daily smoking

The strong negative associations between parental education and the respondent's daily smoking in both genders attenuated when all childhood adversities were adjusted for (Figure 6, Model II; Substudy II, Tables 4 and 5). Further, the inclusion of parental smoking in the model further slightly reduced the associations in women (Substudy II, Table 4). After adjusting for the respondent's own education, the associations lost their statistical significance in both genders (Figure 6; Model IV, Substudy II, Tables 4 and 5). Daily smoking was more common in women who had lived in a single-parent family as a child than in those who had lived with both parents (however, the difference was statistically significant only in the age group 18–24 years, see Table 7). The association lost its statistical significance after all childhood circumstances were adjusted for (Figure 6, Model II).

In age-adjusted models, parental divorce showed statistically significant associations with daily smoking in both genders, as did parental unemployment in men and long-term financial problems, maternal alcohol and mental health problems and serious conflicts within the family in women (Figure 6, Model I). Almost all associations lost their statistical significance after controlling for all childhood conditions. However, in some of them, adjusting for the respondent's own education seemed to reduce the associations further (Figure 6, Model II). The association between daily smoking and parental divorce ( $OR=2.66$ ) remained statistically significant in women even when all indicators relating to current circumstances were controlled for (Figure 6, Model IV).

Daily smoking varied strongly according to parental smoking in both genders. The risk of daily smoking was increased if both the respondents' parents smoked, even if all childhood and current circumstances were adjusted for (Figure 6, Model IV). The effect of a smoking father on men's smoking also remained statistically significant despite all adjustments. In women, the significant association between maternal smoking and the respondent's smoking lost its statistical significance when the effect of the respondent's own education was adjusted for (Figure 6, Model III).

**Figure 6. Daily smoking by childhood circumstances in men and women aged 18–29 years (OR).** Adjusted for age, childhood circumstances<sup>1,2</sup> (parental education, childhood family structure, childhood adversities and parental smoking), the respondent's own education and other current circumstances.



\*  $p < 0.05$

<sup>1</sup> Only statistically significant childhood circumstances ( $p < 0.05$ ) for either gender are presented (for others, see Substudy II, Tables 2, 4 and 5).

<sup>2</sup> Parental primary level education compared to the highest educational category; single-parent family compared to two-parent family; both parents smoking, paternal smoking and maternal smoking compared to those whose neither parent smoked. Adversities compared to those who did not report the specific problem in childhood.



## Heavy drinking

Although parental education was not statistically significantly associated with heavy drinking, there were some apparent associations that did not quite reach statistical significance. For example, women with low parental education seemed to have a lower risk and men with middle parental education a higher risk of being a heavy drinker, even in the model that adjusted for all childhood and current circumstances (Substudy III, Tables 3 and 4).

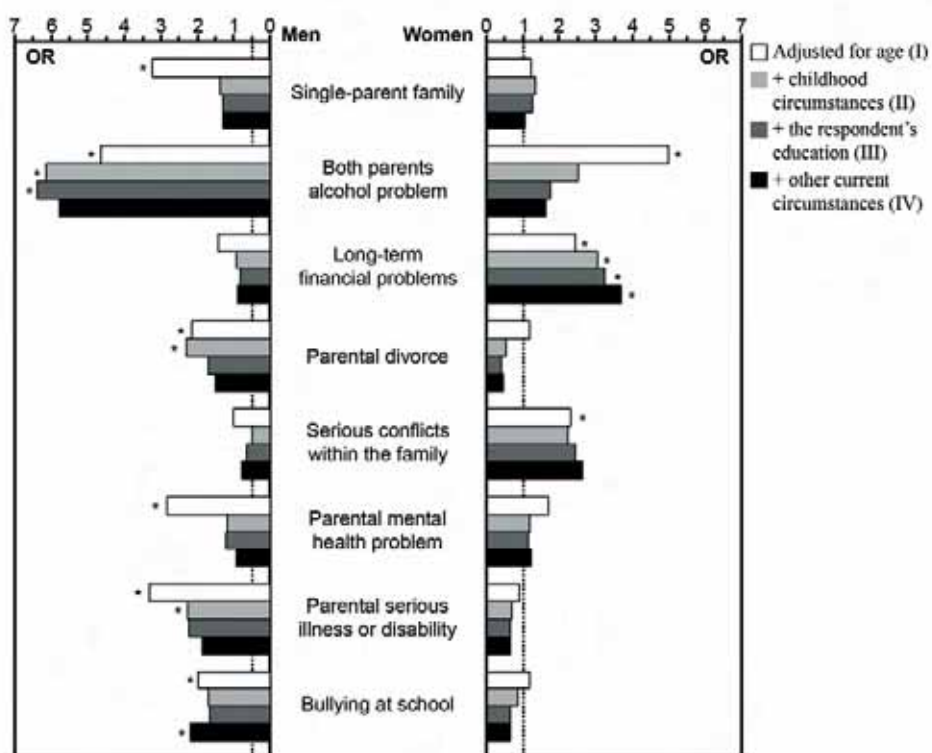
In men, the association between heavy drinking and having lived in a single-parent family in childhood ( $OR=3.23$ ) remained statistically significant when parental education was adjusted for, but disappeared after other childhood adversities were included in the model (Figure 7, Model II; Substudy III, Table 2), mainly because of the effect of parental divorce. The association further decreased slightly after adjusting for the respondent's own education (Figure 7, Model III).

In women, long-term financial problems ( $OR=2.43$ ) and serious conflicts within the childhood family ( $OR=2.31$ ) were associated with heavy drinking. The association between long-term financial problems and heavy drinking remained statistically significant despite all adjustments. In addition, the association between serious conflicts within the family and heavy drinking remained suggestive after controlling for the other explanatory variables (Figure 6, Model IV; Substudy III; Table 3).

In men, parental divorce, parental mental health problem, parental serious illness or disability and having been bullied at school were associated with heavy drinking in the age-adjusted models (Figure 6, Model I; Substudy III, Table 4). However, the associations between heavy drinking and parental divorce as well as parental serious illness or disability disappeared after controlling for the respondent's educational level (Figure 7, Model III). The associations with parental mental health problems and having been bullied at school lost their statistical significance when other childhood circumstances were controlled for (Figure 7, Model II). However, the association with having been bullied at school re-emerged when all factors were adjusted for (Figure 7, Model IV).

Statistically significant associations were found between both parents' alcohol problems and heavy drinking in both genders (Figure 7, Model I; Substudy III, Tables 3 and 4). In women, this association lost its significance when other childhood circumstances were controlled for (Figure 6, Model II). In men, the strong association remained statistically significant even after adjusting for other childhood circumstances, own education and main activity, but after controlling for current family structure and degree of urbanisation of current residence, the association did not quite reach statistical significance (Figure 8, Model IV). However, men whose both parents had an alcohol problem differed from the reference category at the 0.1 significance level in the last model as well (Figure 8, Model IV).

**Figure 7. Heavy drinking by childhood circumstances in men and women aged 18–29 years (OR).** Adjusted for age, childhood circumstances<sup>1,2</sup> (parental education, childhood family structure, childhood adversities and parental alcohol problems), the respondent's own education and other current circumstances (main activity, current family structure and place of residence).



\*  $p < 0.05$

<sup>1</sup> Only childhood circumstances statistically significant for either gender are presented (for others, see Substudy III, Table 2).

<sup>2</sup> Single-parent family compared to two-parent family; parental alcohol problems compared to those whose neither parents had alcohol problems. Adversities compared to those who did not report the specific problem in childhood.

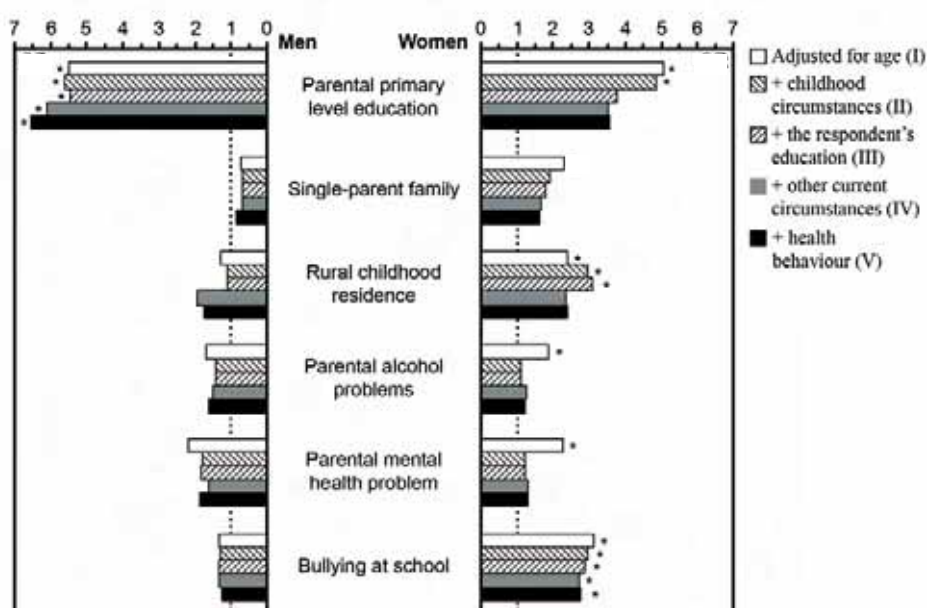
## Overweight and obesity

The clear negative association found in women between parental education and overweight ( $25 \leq \text{BMI} < 30$ ) remained relatively unchanged after all childhood circumstances were included in the model, but lost its statistical significance when adjusting for the respondent's own educational level (Substudy IV, Table 4a). In both genders, the strong association between parental education and obesity ( $\text{BMI} \geq 30$ ) remained strong after all adjustments, although not quite reaching statistical significance

in women in the model adjusted for all current circumstances (Figure 8, Model V; Substudy IV, Tables 4a and 4b). The significant association between primary parental education and obesity in women lost its statistical significance when the respondent's own education was adjusted for (Figure 8, Model III).

The associations of growing up in a single parent family with both overweight and obesity were marginally statistically significant in the age-adjusted model in women (Figure 8, Model I; Substudy IV, Table 4a), but attenuated when parental education was adjusted for (Substudy IV, Table 4a). In men, childhood family structure was not associated with either overweight or obesity (Figure 8; Substudy IV, Table 4b).

**Figure 8. Obesity ( $BMI \geq 30$ ) by childhood circumstances in men and women aged 18–29 years (RRR) compared to normal weight respondents ( $18.5 \leq BMI < 25$ ). Adjusted for age, childhood circumstances<sup>1,2,3</sup>, the respondent's own education, other current circumstances<sup>4</sup> and health behaviour<sup>5</sup>.**



\*  $p < 0.05$

<sup>1</sup> Only childhood circumstances significant for either gender are presented (for others, see Substudy IV, Tables 3, 4a and 4b).

<sup>2</sup> Parental education, childhood family structure and 11 childhood adversities.

<sup>3</sup> Parental primary level education compared to the highest educational category; single-parent family compared to two-parent family; parental alcohol problems compared to those whose neither parents had alcohol problems. Adversities compared to those who did not report the in childhood.

<sup>4</sup> Main activity, current family structure, place of residence.

<sup>5</sup> Smoking, drinking, physical activity, use of vegetables.

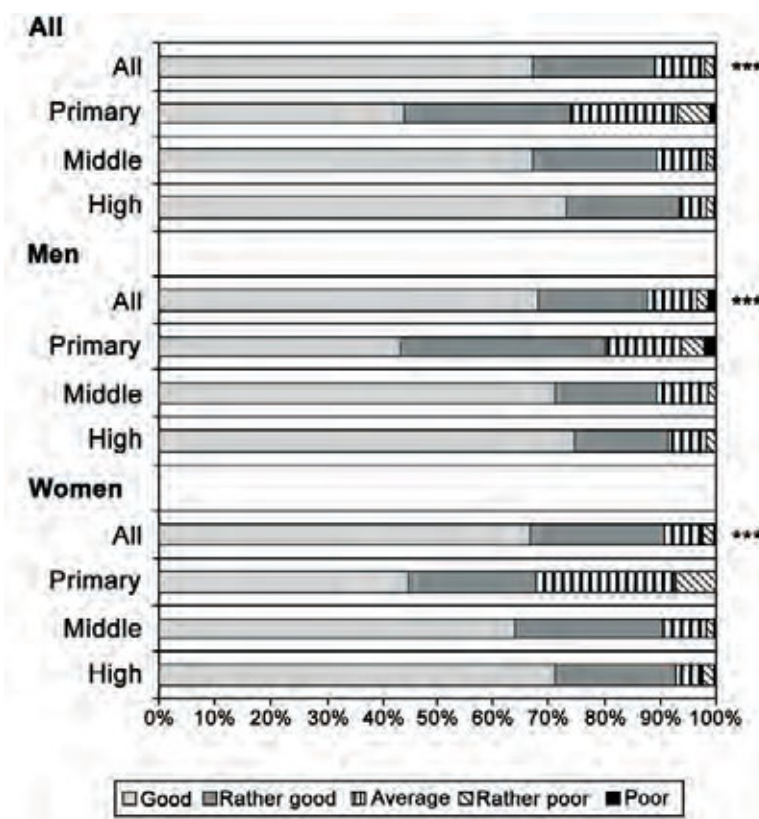
Women who had lived in a rural municipality in childhood were more likely to be obese than those from semi-urban or urban municipalities (Figure 8, Model I; Substudy IV, Table 4a). When current residence and main economic activity were included in the model, the association was no longer statistically significant (Figure 8, Model IV), but remained suggestive despite the rest of the adjustments (Figure 8, Model V). For men, childhood residence was not statistically significantly associated with overweight or obesity (Figure 8). In women, regular parental unemployment was statistically significantly associated with overweight (RRR=2.09) (Substudy IV, Table 4a), whereas parental alcohol and mental health problems and being bullied at school were associated with obesity in the age-adjusted model (Figure 8, Model I; Substudy IV, Table 4a). In addition, serious conflicts within the family were associated with obesity at the  $p < 0.1$  level. Controlling for other childhood factors decreased the RRRs (Figure 8, Model II), and only that of being bullied at school remained a statistically significant predictor of obesity, despite all adjustments (Figure 8, Model V). In men, none of the childhood adversities was statistically significantly associated with overweight or obesity.

## **6.4 Explanatory effects of childhood circumstances, current circumstances and health behaviour on educational health differences (V)**

For the analysis of the explanatory effects of childhood circumstances, current circumstances and health behaviour on educational differences in poor SRH, men and women were analysed together. In total, 70% of young adults aged 18–29 years rated their health as good; 20% reported average, 8% rather poor and only less than 2% poor health (Figure 9). There was no statistically significant gender difference in SRH ( $p = 0.449$ ).

There was a clear gradient in poor SRH according to the respondent's educational level in both genders: the lower the level of education, the more likely it was for the respondent to report average or poorer health ( $p < 0.001$ ). Seven per cent of those in the highest educational category reported average or poorer health, while the corresponding figures were 10% in the middle and 26% in the lowest educational category (Figure 9). In the lowest educational category the likelihood of reporting poor health was almost five times as high as in the highest category. The interaction between educational level and gender in the age-adjusted model was not statistically significant ( $p = 0.215$ ).

**Figure 9. Distribution (%) of self-rated health (SRH) by level of education.**  
*All young adults aged 18–29 years, and separately in men and women. Statistical difference between educational groups, \*\*\*  $p < 0.001$ .*



#### 6.4.1 Associations of childhood circumstances, current circumstances and health behaviour with poor SRH and level of education

Parental education was associated with poor SRH and the respondent's own educational level (Table 8; Substudy V, Table 1): those whose parents had a secondary degree education were the least likely to report poor health. Parental education was also strongly associated with the respondent's own educational level: the higher the level of parental education, the more likely it was for the respondent to have a higher education.

Living in a single-parent family in childhood increased the risk of poor SRH: compared to those who had two parents, the risk of poor SRH was more than twice as high (OR=2.24). Living in a single parent family in childhood also increased the risk of belonging to the lowest educational category: 15% of the lowest-educated came from a single-parent family background as compared to 7% in both the high and middle educational categories. The degree of urbanisation of childhood residence was associated with the respondent's education, as those with a high level of education were most likely to live in urban municipalities. The associations with poor SRH were weaker and rather inconsistent (Table 8; Substudy V, Table 1).

The following childhood adversities predicted poor SRH: long-term financial problems (OR=2.09), parental divorce (OR=1.65), serious conflicts within the family (OR=2.42), parental mental health problems (OR=1.80), parental alcohol problems (OR=1.66), own serious or chronic illness (OR=4.80), parental serious illness or disability (OR=2.55) and being bullied at school (OR=2.89). Regular parental unemployment was not associated with poor SRH. Serious conflicts within the family, parental mental health problems and the respondent's own serious or chronic illness were not associated with the respondent's own educational level. All other childhood adversities that predicted poor SRH were also associated with the respondent's education at the  $p < 0.25$  significance level (Substudy V, Table 1).

Unemployed and laid off respondents had the highest risk of poor SRH (OR=2.89 compared to the full-time or part-time employed,  $p < 0.001$ ) (Table 8; Substudy V, Table 1). Being unemployed was also associated with low educational level: 26% in the lowest, 10% in the middle and 3% in the highest educational category were unemployed or laid off. Degree of urbanisation of current residence was associated with both poor SRH and low education: those in big cities were most likely to report poor health. The interaction between gender and current residence was statistically significant ( $p = 0.048$ ), and the association between current residence and poor SRH was stronger in women than in men (Table 8; Substudy V, Table 1). A larger proportion of the lowest educated (24%) than the highest educated respondents (11%) lived in rural municipalities. Current family structure and having children were associated with the respondent's education, but not with poor SRH.

Daily smokers (OR=2.32), heavy drinkers (OR=2.63), physically inactive (OR=3.18 compared to the physically most active category), obese respondents (OR=2.65) and those who did not eat vegetables frequently (OR=1.80 compared to daily users) were statistically significantly more likely to report poor health than those in the reference categories. All health behaviour factors were associated with the respondent's educational level as those in the lowest educational category had the most health-damaging behaviours (Table 8; Substudy V, Table 1).

**Table 8.** *Distribution (%) of childhood and current circumstances and health behaviours by level of education and their associations (OR) with poor SRH in women and men aged 18–29 years. N= 1,282.*

Explanatory factor	Level of education					Poor self-rated health		
	All, %	High, %	Middle, %	Primary, %	p <sup>1</sup>	OR	p <sup>2</sup>	p <sup>3</sup>
<b>Gender</b>								
Women	53	61	45	47		1.00		
Men	47	39	55	53	0.000	1.15	0.477	
<b>Age</b>								
18–23	61	57	65	60		1.00		
24–29	39	43	35	40	0.032	1.17	0.434	0.166
<b>CHILDHOOD CIRCUMSTANCES</b>								
<b>Parental education</b>								
Secondary	25	37	16	5		1.00		
Intermediate	24	27	21	21		2.01**		
Primary and some vocational	30	22	35	43		1.42		
Primary only	18	11	25	22		1.84*		
Don't know or did not have parents	3	2	2	8	0.000	1.64	0.149	0.866
<b>Childhood family structure</b>								
Two parents	92	93	93	81		1.00		
One parent	8	7	7	15		2.24**		
Other	0.6	0.5	0.2	3	0.000	3.14	0.008	0.053* <sup>4</sup>
<b>Childhood residence</b>								
Urban municipalities	54	58	48	52		1.00		
Semi-urban municipalities	18	17	20	17		0.73		
Rural municipalities	27	23	31	28		1.24		
Abroad	1	1	1	2	0.039	0.37	0.239	0.134
<b>Childhood adversities (yes)</b>								
Long-term financial problems	17	16	17	23	0.235	2.09**	0.001	0.602
Regular parental unemployment	11	8	15	11	0.006	1.31	0.349	0.981
Parental divorce	20	17	21	37	0.000	1.65**	0.022	0.505
Serious conflicts within the family	24	25	23	28	0.484	2.42**	0.000	0.476
Parental mental health problem	8	7	7	10	0.664	1.80*	0.064	0.152
Parental alcohol problem	20	18	21	30	0.026	1.66**	0.026	0.113
Own serious or chronic illness	4	3	3	6	0.328	4.80**	0.000	0.250
Parental serious illness or disability	14	12	15	20	0.075	2.55**	0.000	0.854
Being bullied at school	25	22	25	35	0.024	2.89**	0.000	0.377
<b>CURRENT CIRCUMSTANCES</b>								
<b>Main activity</b>								
Full-time of part-time employed	60	58	64	48		1.00		
Student	22	33	13	1		1.07		
Unemployed or laid off	8	3	10	26		2.89**		
Other	10	5	13	25	0.000	1.52	0.001	0.449
<b>Current family structure</b>								
Married or cohabiting	53	57	48	51		1.00		
Living alone	26	32	21	16		1.32		
Living with own parents or other	21	11	30	32	0.000	1.20	0.460	0.254
<b>Current residence</b>								
Big city	44	53	33	44		1.00		
Urban or semi-urban	39	36	44	32		0.67*		
Rural	17	11	22	24	0.000	0.65	0.108	0.048** <sup>5</sup>
<b>Having children (yes)</b>	21	15	25	35	0.000	1.16	0.527	0.137
<b>HEALTH BEHAVIOUR</b>								
<b>Daily smoking</b>	27	18	32	52	0.000	2.32**	0.000	0.204
<b>Heavy drinking</b>	6	4	6	25	0.000	2.63**	0.001	0.808
<b>Obesity</b>	7	5	8	11	0.035	2.69**	0.002	0.514
<b>Leisure time physical activity</b>								
4 times or more a week	29	31	26	35		1.00		
1–3 times a week	54	57	52	42		1.09		
Less than once a week	17	12	21	23	0.000	3.18**	0.000	0.278
<b>Use of vegetables</b>								
6–7 days a week	52	62	44	34		1.00		
3–5 days a week	27	24	30	31		1.16		
Less than 3 days a week	20	13	26	35	0.000	1.80**	0.031	0.335

\* p<0.1, \*\*p<0.05

<sup>1</sup> Significance of the difference between educational level and explanatory factor, Chi2-test.

<sup>2</sup> Significance of the difference between explanatory factor and poor self-rated health, Wald test p-value within the group.

<sup>3</sup> Significance of the interaction between explanatory factor and gender, Wald test p-value.

<sup>4</sup> Interaction gender\*childhood family structure p=0.053. The association between family structure and poor SRH is stronger in men than in women, p=0.000 and p=0.102, respectively.

<sup>5</sup> Interaction gender\*current residence, p=0.048. The association between residence and poor SRH is stronger in women than in men, p=0.009 and p=0.863, respectively.



#### 6.4.2 Explanatory effects of childhood circumstances, current circumstances and health behaviour on educational health differences

The effect of parental education on the educational differences in poor SRH was weak (Substudy V, Table 2). The effect of childhood family structure was stronger: it explained almost one-tenth of the differences in poor SRH between the highest and the lowest educational category. However, adjusting for childhood family structure reduced the educational health differences only in men (Substudy V, Table 2).

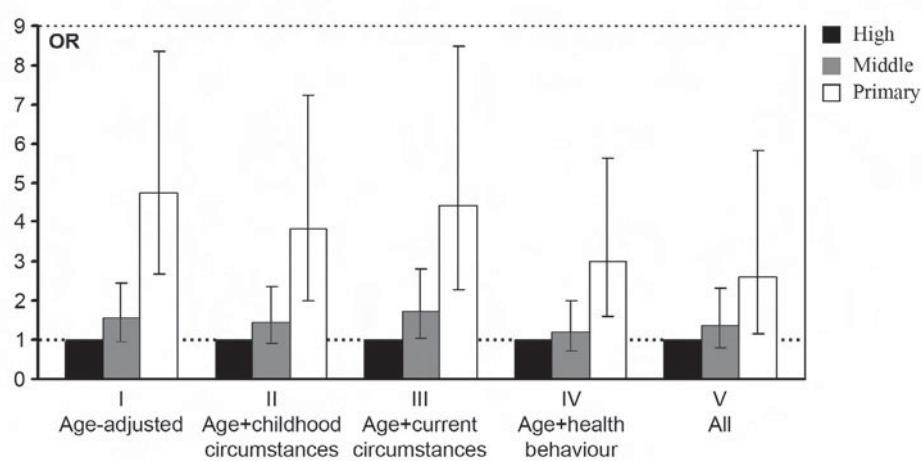
Parental divorce and being bullied at school had strong explanatory effects on educational differences in poor SRH (Substudy V, Table 2), reducing the difference between the highest and the lowest educational category by 11%. For the middle educational category, being bullied at school (16%), parental serious illness or disability (16%) and parental alcohol problems (13%) reduced the ORs the most. All childhood circumstances together explained almost one-fifth (18%) of the difference in poor SRH between the highest and the middle educational category and one-quarter (24%) of the difference between the highest and the lowest educational category (Figure 10, Model II; Substudy V, Table 2).

Adjusting for the respondent's main activity attenuated the difference in poor SRH between the highest and the lowest educational category by 14%. Adjusting for the degree of urbanisation of current residence, on the other hand, accentuated the educational differences, but only in men (Substudy V, Table 2). Together these two factors explained 8% of the health differences between the highest and the lowest educational category (Figure 10, Model III; Substudy V, Table 2).

Daily smoking (24%) and heavy drinking (15%) greatly reduced the differences in poor SRH between the highest and the lowest educational category. Obesity (11%), use of vegetables (10%) and physical activity (8%) also reduced the differences. Furthermore, almost one-third of the health differences between the highest and the middle educational category was explained by both physical activity (30%) and daily smoking (27%) (Substudy V, Table 2). All these behavioural factors together explained 46% of the difference in poor SRH between the highest and the lowest, and 61% of the difference between the highest and the middle educational category (Figure 10, Model IV; Substudy V, Table 2).



**Figure 10. Age- and gender-adjusted educational differences in poor SRH. Adjusted for childhood circumstances<sup>1</sup>, current circumstances<sup>2</sup>, indicators of health behaviour<sup>3</sup> and all. Odds ratios (OR) with 95% CI:s.**



<sup>1</sup> Parental education, childhood family structure, childhood residence, financial problems in the family, parental divorce, parental alcohol problems, parental serious disease or disability and being bullied at school.

<sup>2</sup> Main activity and current place of residence.

<sup>3</sup> Daily smoking, heavy drinking, obesity, physical activity and use of vegetables.

### 6.4.3 The contribution of health behaviour and current living conditions in the effect of childhood circumstances on educational health differences

The contribution of childhood circumstances to the differences in poor SRH between the highest and the lowest educational category was 24%. However, it was assumed that this effect was at least partially shared with the effect of later circumstances and behavioural factors. Thus, the shared explanatory effects and proportion shared with these factors were calculated (Table 9; Substudy V, Table 3).

First, the shared explanatory effect of childhood and current circumstances was four per cent, indicating that 17% of the effect of childhood social circumstances was shared with the effect of later circumstances, mainly employment (as main activity was the one circumstances that reduced educational differences in the first place). Second, the shared effect of behavioural factors and childhood circumstances was 15%, indicating that the proportion of the effect of childhood circumstances shared with them was 63%. Thus, nearly two-thirds of the contribution of childhood circumstances was shared with behavioural factors, and just under one-fifth with current circumstances (Table 9; Substudy V, Table 3).

**Table 9.** *Age- and gender-adjusted differences in poor SRH between the high and the primary educational categories<sup>1</sup>. Adjusted for childhood circumstances, current circumstances and behavioural factors. Odds ratios (OR) with 95% CI:s and percentage reductions (%). Shared effects of behavioural factors/current circumstances and childhood circumstances (%) and the proportion (%) of the effect of childhood circumstances shared with current circumstances/behavioural factors.*

Adjusted factors	Educational level		% reduction in OR <sup>2</sup>	Proportion shared %
	High	Primary		
		<b>OR [95 CI]</b>		
Base model <sup>3</sup>	1.00	4.69 [2.63–8.32]		
Childhood circumstances	1.00	3.80 [2.00–7.23]	24	
Current circumstances	1.00	4.41 [2.29–8.49]	8	
Behavioural factors	1.00	3.00 [1.60–5.61]	46	
Childhood and current circumstances	1.00	3.64 [1.78–7.41]	28	
Childhood circumstances and behavioural factors	1.00	2.65 [1.72–2.02]	55	
All	1.00	2.61 [1.17–5.83]	56	
Shared effect of current and childhood circumstances (%)			(24+8)-28=4	
Shared effect of behavioural factors and childhood circumstances (%)			(24+46)-55=15	
Proportion of the effect of childhood circumstances shared with current circumstances (%)				4/24=17
Proportion of the effect of childhood circumstances shared with behavioural factors (%)				15/24=63

<sup>1</sup> Reduction % and shared effects not calculated for the middle educational category as it did not differ statistically significantly from the reference category in the base model.

<sup>2</sup> Reduction % was calculated:  $[(OR_{(base\ model)} - OR_{(base\ model + intermediated\ factors)}) / (OR_{(base\ model)} - 1)]$

<sup>3</sup> Adjusted for age and gender.

## 7 DISCUSSION

### 7.1 Main findings and their discussion

There are four main findings in this thesis. The *first* main finding suggests that childhood circumstances predict the health of young adults. Many childhood adversities were found to be associated with poor SRH and psychological distress in early adulthood. However, fewer associations were found with somatic morbidity. The effects seemed to be more or less independent of the young adult's own education. It seems that the educational path has only a minor mediating role in the effect of childhood circumstances on health indicators in young adults in Finland. However, the young adult's own education has a strong independent effect on poor self-rated health.

The *second* main finding suggests that childhood circumstances have a strong effect on smoking and heavy drinking in early adulthood, and that youth paths and the educational path in particular play a role in mediating this effect. Most notably, parental smoking and alcohol abuse have an influence on the corresponding behaviours in offspring.

The *third* main finding emphasises the strong effect of parental education on early adult obesity independently of the young adult's own educational level. In general, childhood circumstances have a role in the development of both overweight and obesity, more so in the case of obesity than overweight and more clearly in women than in men.

The *fourth* main finding is that there are marked educational differences in SRH in early adulthood and that childhood social circumstances explain a substantial part of them. The effect of childhood circumstances is largely shared with the effect of health behaviours adopted by early adulthood. Smoking and heavy drinking contribute substantially to these educational health differences.

According to the results of this thesis, childhood circumstances have direct effects on health outcomes and both direct and mediated effects on the three indicators of health behaviour in early adulthood. It seems then that the pathways from childhood social environment to early adult physical and mental health do not operate through the respondent's own educational level, but have independent effects. The other potential pathways will require further research. However, the thesis indicates that there are pathways from childhood and youth to adult health behaviour where education plays an important mediating role. These pathways may indicate an accumulation of problems, as those who come from less favourable childhood backgrounds are more likely to attain lower educational levels, develop more health-damaging lifestyles and consequently have poorer health.

### 7.1.1 From childhood circumstances to early adult health

According to the results of this thesis, almost all of the childhood adversities studied were associated with poor SRH and psychological distress. In line with previous studies (Lundberg 1997; Bosma, van de Mheen et al. 1999; Dube, Felitti et al. 2003; Huurre, Aro et al. 2003), better childhood circumstances were found to be associated with better health.

Parental education was here found to have only a minor and rather inconsistent effect on poor self-rated health and on psychological distress in young Finnish adults, even though it has earlier been reported that low primary SEP is associated with poor adult health (Rahkonen, Arber et al. 1995). In addition, a Finnish follow-up study (TAM) found that parental SEP had an effect on early adult well-being, as women from a manual class of origin had more distress symptoms than those from a non-manual background. However, no association was found with physical health (Huurre, Aro et al. 2003). Although the effect of parental education was weak in this thesis, long-term financial problems in childhood had independent effects on both poor SRH and psychological distress in men (and a significant age-adjusted association in women), suggesting that economic hardship in childhood may have an independent effect on health in early adulthood. It is likely that parental education also reflects non-material aspects of SEP in addition to financial hardship. Other possible reasons for the differences between these and earlier results may also be due to methodological differences, the use of different SEP measures, or the fact that the cohort of young adults lived their childhood and youth in different socio-cultural contexts where parental education may have been differently distributed and may have had different effects.

However, although the effect of parental education on the health outcomes was rather weak, the predictive effect of many childhood adversities was clear, especially on poor SRH and psychological distress. Growing up in a single-parent family increased the risk of both poor self-rated health and psychological distress in men. Previous studies have also found that growing up in a single-parent family has disadvantages to health: a Swedish study reported that children of single parents had an increased risk of severe morbidity (Ringsbäck-Weitoft, Hjern et al. 2003), and a previous Finnish study has found that living in a single-parent family increases the risk of mortality in middle adulthood (Pensola 2004). According to the findings of this study, men from single-parent families were found to be more likely to have psychological distress than those from families with two parents. Earlier studies have also reported a higher occurrence of depressive and anxiety disorders in persons from single-parent families (Mäkikyrö, Sauvola et al. 1998; Barrett and Turner 2005; Fergusson, Boden et al. 2007). For example, a US-based study on a large sample of young adults found higher levels of depressive symptoms in those who came from stepfamilies, single-parent

families and single-parent families with another relative present compared to mother-father families (Barrett and Turner 2005). Another recent study suggested that the associations between exposure to single parenthood in childhood and mental health in young adulthood was explained by the social and contextual factors associated with exposure to single parenthood (Fergusson, Boden et al. 2007). The finding regarding greater impact of a single-parent family background on men's health is interesting and certainly warrants further research.

The effects of living in a single-parent family and parental divorce obviously overlap to some extent, although they described different phases of the life-course in this thesis (parental divorce prior to age 16 and living in a single-parent family at age 7). Nevertheless, parental divorce was here found to have a predictive effect on psychological distress in men, and the effect was suggestive in women as well. Parental divorce seems to be an important risk factor for poor mental health later in life, particularly in women (Wadsworth, Maclean et al. 1990; Rodgers 1994; Palosaari and Aro 1995; Palosaari, Aro et al. 1996; Rodgers, Power et al. 1997; Huurre, Junkkari et al. 2006). A recent Finnish study found that women from divorced as compared to non-divorced families reported more psychological symptoms. However, this difference was not found in men (Huurre, Junkkari et al. 2006). The predictive effect of parental divorce was attenuated by other childhood circumstances. Education did not mediate the effect of parental divorce on early adult psychological distress, but potential confounding childhood adversities and psychological factors are likely to play roles in the process, as suggested in previous studies. The mediating factors between childhood experience of parental divorce and subsequent depression in early adulthood were assessed in another Finnish study and in women, the long-term impact of parental divorce was found to be mediated via low self-esteem and lack of closeness to father (Palosaari, Aro et al. 1996). In another long-term study of the effects of divorce, children in divorced families revealed more stressful paths and more distress in early adulthood (Aro and Palosaari 1992).

Parental mental health and alcohol problems and serious conflicts within the family were found to increase the risk of psychological distress and poor SRH in both genders. According to previous findings, parental depressive disorders are known to raise the risk of mental disorder in offspring (Mäkikyrö, Sauvola et al. 1998; Marmorstein, Malone et al. 2004; Rosenman and Rodgers 2006). Parental alcohol and mental health problems are risk factors for depression and depressive disorders later in life, although their effect may vary by gender. In a Finnish study on the adult population, paternal mental health problems showed a particularly strong association with depressive disorders in men and maternal mental health problems with depressive disorders in women. Maternal alcohol problems, however, were associated with alcohol use disorders in both genders (Pirkola, Isometsä et al. 2005). There is some indication that

depression in adult children of alcoholic parents may largely result from the greater likelihood of adverse childhood experiences in a home with alcohol-abusing parents (Anda, Whitfield et al. 2002). In a Swedish study by Lundberg from the early 1990s, it was found that conflicts in the family during upbringing were strongly related to illness later in life. Having a broken family and, to some extent, economic hardship during childhood were clearly associated with illness later in life. This finding did not change even after controlling for age, gender and paternal SEP (Lundberg 1993).

Besides familial social factors, having been bullied at school predicted both psychological distress and poor SRH in young Finnish adults. Although the impact of school context was not examined in detail in this study, it is an important life sphere in childhood and adolescence and experiences there may have a prominent role in the development of poor health and mental disorders decades later. Being bullied at school has been found to predict a variety of adult mental disorders in both genders in the Finnish adult population (Pirkola, Isometsä et al. 2005). Other school difficulties, such as problems related to learning and behavioural difficulties, have also been found to be associated with poor SRH in early adulthood (Lavikainen, Koskinen et al. 2006).

A large number of studies have shown that many common somatic disorders depend on social factors, at least in middle-aged and elderly persons (Lahelma and Rahkonen 1997; Power and Matthews 1997; Mackenbach, Bos et al. 2003). The lack of such clear associations in young adults in this study is most likely due to the very different physical disease spectrum in early adulthood as compared to older age groups, on which many of the previous studies are based. In particular, lifestyle-determined common chronic conditions (such as cardiovascular diseases and chronic bronchitis) are practically non-existent in young adults and these diseases in particular have been shown to be associated with SEP in later life. Furthermore, it has been suggested that some increasing disease groups such as allergies and asthma that are common in the younger population, are more prevalent in higher SEP groups (Basagana, Sunyer et al. 2004). However, in this study it was found that low maternal education had an independent effect on somatic morbidity in young adult women, as did own serious or chronic illness and bullying at school in both genders. This emphasises continuity of somatic problems from childhood and youth to early adulthood.

#### Independent effect of childhood circumstances on adult health outcomes

The results of this thesis lend support to the independent effects of childhood circumstances on early adult health. The effect of childhood circumstances seems to be rather independent of the respondent's own education. Some previous studies have also found that childhood circumstances and parental SEP have an effect on adult health independently of adult SEP (van de Mheen, Stronks et al. 1998; Huurre, Aro

et al. 2003). Some studies have indicated that adverse SEP in childhood is associated with poorer health independently of adult SEP and across diverse measures of disease risk and physical functioning (Power, Atherton et al. 2007). A study based on the 1958 British birth cohort also discovered that the effects of childhood circumstances on poor SRH were not removed by the inclusion of contemporary factors (Hertzman, Power et al. 2001). However, there is also strong evidence that parental SEP is associated with youth paths (Power and Hertzman 1997; van de Mheen, Stronks et al. 1997; Pensola and Martikainen 2004), which in turn affect health through causation mechanisms. Besides SEP, specific adversities may affect the educational paths. For example, emotional disruption in the family can reduce the child's likelihood of high educational attainment. Parental divorce and separation have also been shown to be associated with reduced educational attainment (Ely, Richards et al. 1999).

In addition to educational pathways, there are some other important pathways by which childhood circumstances may influence early adult health, although they were not analysed in the first Substudy of this thesis. The effect of childhood circumstances on adult health, independent of adult SEP, may partly operate through unhealthy behaviour (van de Mheen, Stronks et al. 1998) as it has been found to mediate the association between parental SEP and adult disease risk (Pensola and Valkonen 2000). Those from unfavourable childhood circumstances may be more likely to adopt unhealthy behavioural patterns, which may result in poor health by early adulthood. For example, unfavourable circumstances may increase the risk of early initiation of smoking or heavy drinking, which greatly enhance the risk of poor self-rated health in early adulthood. Secondly, it is also necessary to consider the importance of psychological mechanisms in the examination of the negative effects of adverse socioeconomic conditions in childhood. In a Dutch study from the late 1990s, a higher prevalence of negative personality profiles and adverse coping styles in subjects who grew up in lower social classes explained part of the association between low primary SEP and poor adult health (Bosma, van de Mheen et al. 1999). In this thesis the effect of employment and family formation paths were not examined as mediators in relation to health outcomes, even though earlier studies have drawn attention to their potential effect.

### 7.1.2 Pathways to smoking and heavy drinking in early adulthood

The *second* main finding of the study suggests that childhood circumstances have a strong effect on smoking and heavy drinking in early adulthood and that current circumstances, and education in particular, play a role in mediating this effect. It has been suggested that the reasons why an individual adopts one form of behaviour



instead of another include the influences of earlier life experiences and the current social and economic environment (Droomers 2002).

According to this thesis, parental smoking and the individual's own education were the strongest determinants of daily smoking in early adulthood. In women, parental divorce, current family structure and current physical activity were also associated with daily smoking. A variety of childhood adversities predicted heavy drinking as well, and the impact was partly independent, but also mediated through other childhood and adult circumstances. In addition, these mechanisms were somewhat different in men and women. Again, the strongest mediating factor between childhood circumstances and heavy drinking was the respondent's own educational level. The effect of the respondent's low education and unemployment on heavy drinking remained statistically significant after all adjustments, in both genders.

#### Childhood circumstances and smoking and heavy drinking in early adulthood

It has been reported that parental SEP is associated with alcohol use (Droomers, Schrijvers et al. 2003) and smoking (Green, Macintyre et al. 1991; Scarinci, Robinson et al. 2002; Huurre, Aro et al. 2003; Jefferis, Graham et al. 2003; Jefferis, Power et al. 2004; Naidoo, Warm et al. 2004; Droomers, Schrijvers et al. 2005; Fagan, Brook et al. 2005) in adolescence and adulthood, with higher prevalences observed for those with low primary SEP. The results of this thesis confirm that parental education has a strong gradient in daily smoking among young adults. However, parental education affected smoking through different paths. First, lower parental education increased the risk of childhood adversities and parental smoking. The effect of parental education was also mediated through the respondent's own education, which was strongly associated with daily smoking.

Parental education showed no statistically significant association with heavy drinking, but self-reported long-term financial problems in the childhood family did have a strong association with heavy drinking in women, independent of adult circumstances. Low parental SEP has been found to predict heavy alcohol use in adolescence (Droomers, Schrijvers et al. 2003), but some studies have found no association between primary SEP and alcohol use in adolescents (Tuinstra, Groothoff et al. 1998). Children growing up in low socioeconomic households have been found to have a high risk of alcohol dependence at age 26 (Poulton, Caspi et al. 2002). In this thesis, the question of the association between low parental SEP and heavy drinking in early adulthood remains partly unsolved; different aspects of SEP may be differently associated with heavy drinking. In a large Swedish cohort study, SEP was found to decrease but parental wealth to increase the risk of alcoholism in young adult men (Andreasson, Allebeck et al. 1993). The association between childhood SEP and heavy drinking in adulthood



has been explained by the higher prevalence of familial alcohol problems and lower parental attachment in those from lower SEP groups, for example (Droomers, Schrijvers et al. 2003).

Living in a single-parent family was an important determinant of daily smoking in women and heavy drinking in men. The association was partly explained by parental divorce and the fact that those from single-parent families had a lower parental education and more childhood adversities. Parental divorce before the respondent was 16 years of age and living in a single-parent family at the age of seven predicted heavy drinking in men. Corresponding findings have been reported previously (Kuh and Maclean 1990; Hope, Power et al. 1998; Anda, Whitfield et al. 2002). However, the effect of living in a single-parent family became much weaker after adjusting for the experience of parental divorce. Furthermore, the effect attenuated markedly after adjusting for the respondent's own education. It thus seems that the effect of parental divorce is mediated through the respondent's own educational level, as those who had experienced divorce in childhood had a lower education and were more likely to be heavy drinkers. Some studies, however, have found that living with both parents is not associated with reduced levels of drinking, and it has been suggested that living with both parents is a less robust barrier to substance use than qualitative aspects of family life (McArdle, Wieggersma et al. 2002). The psychological and psychosocial effects of parental divorce are probably stronger than the effect of living arrangement as such.

According to the findings of this thesis, parental substance abuse predicted corresponding behaviours in offspring during early adulthood: parental smoking affected daily smoking and parental alcohol problems heavy drinking in offspring as an adult, independently of current circumstances. The background for these findings can be found in the mechanisms of social and biological inheritance (White, Hopper et al. 2003). According to the results of this thesis, the risk of being a daily smoker in early adulthood was the greatest in respondents whose both parents smoked. It has been reported earlier that smoking in one's social environment, parental smoking (Green, Macintyre et al. 1991; Rossow and Rise 1994; White, Pandina et al. 2002; White, Hopper et al. 2003; Barman, Pulkkinen et al. 2004; Fagan, Brook et al. 2005; Brook, Pahl et al. 2006), especially maternal smoking (Kandel, Wu et al. 1994; Kandel 1995) predict smoking, although the findings are partly inconsistent (Avenevoli and Merikangas 2003) and gender-specific (White, Pandina et al. 2002). The reason why parental smoking predisposes children to become daily smokers may lie in at least three mechanisms: firstly, biological vulnerability to tobacco (genetic factors and exposure to nicotine in utero and during childhood may influence both the initiation and maintenance of tobacco use); secondly, 'social heredity' and modelling (in which offspring imitate their parents' behaviour) may influence initiation through adolescents' own perceptions of smoking; and thirdly, tolerant attitudes towards smoking

may influence the young person's capabilities to resist smoking and continuing the habit. Parental attitudes towards smoking and parents' reactions to smoking initiation may play an important role in maintaining smoking through the teen years (Droomers 2002).

The results of this thesis also support earlier findings which suggest that children of parents with alcohol problems are at a higher risk of using excessive amounts of alcohol themselves (Anda, Whitfield et al. 2002; Pirkola, Isometsä et al. 2005), although statistically significant associations were found only in families where both parents had alcohol problems. In men, parental alcohol problems had an independent effect on heavy drinking, whereas in women the effect was confounded by other childhood circumstances. Both alcohol-specific and non-alcohol-specific parenting influences affect the development of alcohol abuse in offspring (Jacob and Johnson 1997). Following the example of parental drinking behaviour, development of alcohol-related attitudes, parent-child relationship, unpredictable home-life and genetic vulnerability, for example, are particularly relevant to the development of alcohol abuse (Zeitlin 1994; Steinhausen 1995; Anda, Whitfield et al. 2002).

#### Education as a mediator and determinant of smoking and heavy drinking

According to this thesis, the impact of childhood circumstances on daily smoking and heavy drinking was partly independent but also confounded by or mediated through other childhood and adult circumstances. In addition, these mechanisms were somewhat different in men and women. The strongest mediating factor was the respondent's own educational level. The effect of the respondent's low level of education on smoking, and in the case of heavy drinking also unemployment, remained statistically significant after all adjustments and in both genders. This indicates that the respondent's educational and employment paths are significant with respect to smoking and drinking.

The research for this thesis revealed marked differences in daily smoking and heavy drinking by the respondent's educational level, with those in the lowest educational group being at the highest risk. Earlier results on SEP differences in alcohol use have been somewhat inconsistent, which may be due to differences in the way its various dimensions (e.g. high total consumption, frequency of drinking, heavy occasional drinking) operate (Casswell, Pledger et al. 2003). It has been reported that heavy drinking is more common in lower than in higher educational groups (Droomers, Schrijvers et al. 1999; O'Donnell, Wardle et al. 2006), but also that women with high SEP consume more alcohol than those with low SEP (Ahlström, Bloomfield et al. 2001). Young adults with a low education have been found to drink significantly more on single drinking occasions, and those with high SEP to drink more frequently

(Casswell, Pledger et al. 2003; Indrei, Carausu et al. 2004). Financial problems also contribute to the educational gradient in drinking (Droomers, Schrijvers et al. 1999). No statistically significant association was found in this thesis between income and heavy drinking, possibly because young adults are still in an unstable stage of life in regard to work and income.

Educational differences in smoking and heavy drinking may emerge through different mechanisms. From the point of view of the causation explanation, a high level of education may be conducive to a healthy lifestyle, and educational differences in heavy drinking and daily smoking may be a result of the unequal distribution of important determinants of drinking in different educational groups. An earlier Finnish study has also found that achieved social position determines health-related behaviours more strongly than class of origin, emphasising the way that education facilitates health values and behaviours as well as future social position (Karvonen, Rimpelä et al. 1999). Based on the selection explanation, however, it is reasonable to assume that those who reach a higher level of education in adulthood have had better health even in childhood and adolescence, while those reaching only a low level of education have had poorer health and a health compromising lifestyle (Wood, Sher et al. 1997; Koivusilta, Rimpelä et al. 1998; Koivusilta, Rimpelä et al. 1999). As for smoking and heavy drinking, it is necessary to take into account the possible effect of (indirect) selection: daily smoking or heavy drinking (or both) may have affected the respondent's educational tracks (Koivusilta, Rimpelä et al. 1998; Koivusilta, Rimpelä et al. 1999) and other living conditions. However, this process may also be influenced by childhood social circumstances: for example, the educational differences observed in heavy drinking may arise from early life circumstances and experiences (Kuh and Maclean 1990), which affect both education and alcohol use, although the results here do not lend much support this notion as the effect of the respondent's own education on heavy drinking was rather independent of childhood circumstances.

#### Other potential life-course factors

There are also other factors in childhood and adolescence that may affect substance abuse in addition to, or in interaction with, familial social factors. Smoking and heavy alcohol use are complex phenomena and they are affected by various factors, many of which were not studied in this thesis. Examples include peer groups, early onset of substance use, youth cultures and school contexts, which influence the initiation and maintenance of smoking (West, Sweeting et al. 1999; White, Hopper et al. 2003) and play a part in the formation of drinking behaviour (Holman, Jensen et al. 1993; Bahr, Marcos et al. 1995; Jones-Webb, Short et al. 1997; Bonomo 2005; D'Amico, Ellickson et al. 2005; Pitkänen, Lyyra et al. 2005). However, in a longitudinal study of school-aged children in six European countries, peer smoking did not emerge as an

important predictor of smoking onset in most countries. The results did, however, lend support to the selection mechanism, indicating that adolescents choose friends with similar smoking behaviour. In addition, support was found for the impact of parents on adolescent behaviour and the choice of friends (de Vries, Candel et al. 2006). Smoking is often initiated during adolescence. At that time the environment plays an important role in daily life (peers, school environment, family environment). After leaving school and moving into adult life, other socioeconomic determinants may have a stronger influence on smoking initiation and continuation.

As regards smoking and drinking, it is necessary to consider not only the social environment but also the role of biological inheritance as a possible explanation, and particularly the interaction between these two. The prevalence of daily smoking in early adulthood, for instance, is determined by the incidence of smoking initiation, maintenance and the quit rate during adolescence. The initiation, maintenance and cessation of smoking are modulated by environmental factors such as childhood experiences, but smoking behaviour has also been shown to be connected with personality traits, the distribution and structure of nicotine receptors and other genetically determined factors (Li 2003; White, Hopper et al. 2003). Several twin and adoption studies have reported that genetic factors explain about 50% of both smoking initiation and smoking persistence (Madden, Heath et al. 1999; Sullivan and Kendler 1999; Li 2003).

### 7.1.3 The effect of parental education on early adult obesity

The *third* main finding of this thesis is that childhood circumstances have a role in the development of overweight and obesity in young Finnish adults, more so in the case of obesity than overweight and more so in women than in men. The results revealed independent childhood predictors of obesity. In particular, they showed that parental education has a strong effect on early adult obesity, even independently of the young adult's own education. A strong inverse association remained between parental education and obesity in both genders even when other childhood circumstances and potentially mediating current circumstances were taken into account. Being bullied at school had an association with obesity in women independent of all other factors. In women, parental alcohol and mental health problems predicted obesity, but these associations attenuated when all childhood circumstances were adjusted for.

In general, overweight was quite weakly associated with childhood circumstances. In women, some childhood circumstances (low parental education, parental unemployment and single-parent family) were associated with overweight, but the effects were largely attenuated by other childhood circumstances. In addition, the effect of parental education was partly mediated by the respondent's own educational

level and other circumstances. In men, no statistically significant childhood predictors of overweight were found. The lack of strong associations may be due to the choice of the cut-off point – as BMI fails to distinguish between lean body and fat mass, it has been suggested that a BMI of 25 does not accurately differentiate overweight from normal weight men (Garn, Leonard et al. 1986). It can be suggested that overweight young adults, particularly men, are a heterogeneous group, whereas obese persons constitute a more distinct group with specific determinants. The effect of childhood may be more apparent when BMI is higher.

#### Mechanisms between primary SEP and adult obesity

The results of this thesis confirm earlier findings regarding the strong effect of parental education on adult obesity. It has been reported that low primary social background has long-term effects on adult overweight and obesity (Power and Moynihan 1988; Kark and Rasmussen 2005; Power, Graham et al. 2005; Crossman, Anne Sullivan et al. 2006). Factors related to disadvantaged social origins seem to increase the risk of obesity, particularly in women: in a study comparing seven population-based studies in six countries, manual origin was found to increase the risk of obesity in women, while the effects were weaker in men (Power, Graham et al. 2005). A recent US study revealed that inequalities in overweight and obesity in young adults were evident by family SEP and even strengthened by their own (Yang, Lynch et al. 2008).

The findings of this thesis indicate that the respondent's own education partly mediates the effect of parental education on obesity, especially in women, but parental education also seems to have an independent effect. A previous Finnish study found that women from a manual class of origin had higher rates of overweight and higher BMI than those in other groups. Controlling for the person's own SEP, the effect of parental SEP diminished but remained significant in women up to 32 years of age (Huurre, Aro et al. 2003). Earlier studies have found that educational attainment is an important mediating factor in the relationship between socioeconomic adversity in childhood and being overweight in adulthood (Lawlor, Batty et al. 2005). However, other studies have observed that childhood social origin has an effect on early adult obesity independent of later SEP (Parsons, Power et al. 1999; Poulton, Caspi et al. 2002); this is supported by the results of this study. Power and Moynihan found that children from manual backgrounds were more likely to become overweight and obese young adults and to remain overweight or obese compared with their non-manual peers (Power and Moynihan 1988). In young adult Swedish men, the prevalence of overweight and obesity were higher in those with low-educated than in those with high-educated mothers (Kark and Rasmussen 2005). The association between one's primary social class and obesity is probably confounded by parental obesity as well (Wada and Ueda 1990), which too few studies have been able to take into

account (Parsons, Power et al. 1999). A study from northwest Germany revealed that overweight parents of low SEP have a high risk of overweight and obese children (Danielzik, Czerwinski-Mast et al. 2004). One possible explanation is that even in adolescence, those from households with a higher educational level seem to have eating patterns that are healthier and closer to dietary recommendations (Anderson, Macintyre et al. 1994; Hamasha, Warren et al. 2006). Social background has been found to have an even stronger effect on young people's daily diet than on other lifestyles (Roos, Karvonen et al. 2004).

There are several possible explanations for the association between childhood SEP and obesity (independent of later SEP). Childhood environment itself may have long-term impact on obesity through, for example, nutrition in infancy (undernutrition, overnutrition) (Power and Parsons 2000), psychological and social factors (e.g. emotional deprivation) (Kaplan and Kaplan 1957; French, Story et al. 1995; French, Perry et al. 1996) and social and cultural norms (attitudes and restraints) (Jeffery, French et al. 1991; Wardle, Volz et al. 1995; Jeffery 1996; Jeffery and French 1996). SEP differences in cultural and social norms provide a potential explanation for individuals from a manual background, who are more likely than those of non-manual origin to both become overweight and to maintain their overweight through adolescence to early adulthood (Power and Moynihan 1988). For instance, dieting is more common in women with higher SEP (Jeffery, French et al. 1991), and the desire to be thin and the tendency to appreciate slimness is greater in schoolgirls with higher SEP (Wardle, Volz et al. 1995). In general, healthy dietary habits and physical activity adopted already in childhood and adolescence may have long-term impacts; for example in a Finnish longitudinal study (LASERI), it was found that maintaining a high level of physical activity from youth to adulthood was independently associated with a lower risk of abdominal obesity in women (Yang, Telama et al. 2006).

Furthermore, factors related to emotional development in early life may affect the development of obesity (Lissau and Sørensen 1994). The early psychosomatic theory of obesity (Kaplan and Kaplan 1957) suggests that there is a link between childhood experiences and eating and comfort. Some hypotheses refer to self-esteem as a factor contributing to the development of obesity, but the evidence is inconsistent (French, Story et al. 1995). In this thesis it was found that childhood adversities, such as parental mental health and alcohol problems, were associated with obesity in women, but their effect disappeared after adjusting for other childhood circumstances. Experienced in childhood, parental neglect (Lissau and Sørensen 1994), self-reported sexual and non-sexual abuse (Felitti 1993; Williamson, Thompson et al. 2002) and parental alcoholism (Felitti 1993) have been found to be associated with adulthood obesity. In a Danish longitudinal study, parental neglect during childhood was associated with a high risk of obesity in early adulthood, independent of age, childhood BMI and

social background (Lissau and Sørensen 1994). Psychological characteristics formed throughout childhood may have a long-term impact on obesity (Parsons, Power et al. 1999). Childhood social circumstances and problems are associated with later overweight and obesity (Johnson, Cohen et al. 2002; Bachman 2004).

#### Other childhood circumstances as predictors of obesity in adulthood

According to the results of this thesis, being bullied at school had an independent effect on obesity in women. This finding is probably confounded by the fact that bullied children and adolescents may already be obese at school and are bullied partly for that very reason. It has been suggested that childhood obesity is a strong predictor of obesity in early adulthood (Magarey, Daniels et al. 2003). Previously, it has been reported that other factors related to the school environment, such as difficulties at school are associated with adult obesity (Lissau and Sørensen 1993).

The results of this thesis indicate that other childhood adversities also predict overweight and obesity. In women, parental alcohol and mental health problems were found to predict obesity, but these associations attenuated when all childhood circumstances were adjusted for. The stronger impact of childhood circumstances on women's obesity may reflect differences in vulnerability to these adversities. Overweight was quite weakly associated with childhood circumstances. Again in women, childhood circumstances (low parental education, parental unemployment and single-parent family) were associated with overweight, but the effects were largely attenuated by other childhood circumstances.

#### Educational differences in overweight and obesity in men and women

Some indications were found of a relationship between the respondent's own educational level and both overweight and obesity in women. However, in men the educational differences were almost non-existent. Previous studies have reported a clear and consistent inverse relationship between SEP and obesity in adulthood in women, but the relationship has been inconsistent for men and children (Sobal and Stunkard 1989; McLaren 2007). In the USA, socioeconomic inequalities in overweight and obesity in early adulthood were found only among women (Yang, Lynch et al. 2008). Cross-sectional data provide no information on the existence and direction of causality between education and obesity (Stunkard and Sørensen 1993). Even when an apparent cross-sectional relationship between obesity and education is found, a reverse causation is probably involved: obesity leads to downward social mobility (Gortmaker, Must et al. 1993; Laitinen, Power et al. 2002). In this case the relationship between low SEP and adulthood obesity may be confounded by childhood or adolescent obesity



(Magarey, Daniels et al. 2003), which could not be controlled for in this study. It has been suggested that childhood overweight is significantly associated with severe obesity in women and men, the effect being stronger for men. This demonstrates the importance of childhood overweight as a risk factor for severe adult obesity over the life-course (Whitaker, Wright et al. 1997; Williams, Davie et al. 1999; Ferraro, Thorpe et al. 2003). Other studies have also emphasised the association between childhood overweight and later obesity: high normal weight in childhood has been found to predict becoming an overweight or obese adult (Field, Cook et al. 2005).

The inverse relationship between SEP and obesity may also be explained by differences in health behaviours (Jeffery, French et al. 1991). Women with higher SEP are more likely to control their weight and report healthier dietary habits and more regular physical activity (Jeffery, French et al. 1991; Wardle and Griffith 2001). Although the association between health behaviour and obesity has been established in several studies, the explanatory effect of contemporary physical activity and use of vegetables was rather weak in this thesis, as was the use of sweet drinks and sweets, current smoking and alcohol use. This finding may have been affected by general problems in the measurement of health behaviour (Seidell 1998), and food behaviour in particular. In this study, there was some indication that childhood circumstances may affect educational differences in obesity.

#### 7.1.4 Educational health differences and their determinants in early adulthood

The *fourth* main finding of this thesis is that there are marked educational differences in self-rated health in early adulthood and that these differences are to a great extent explained by childhood social circumstances. The effect of childhood circumstances was largely shared with the effect of health behaviours adopted by early adulthood and also with the effect of the respondent's employment status. Daily smoking and heavy drinking were strongly associated with educational health differences. The results strengthen the assumption that educational health differences in adulthood result from factors operating at different stages of the life-course (Power, Matthews et al. 1998) and these factors are in fact related to each other.

##### Educational differences in self-rated health in early adulthood

This study revealed marked educational differences in poor SRH in early adulthood in young Finnish adults. After a period of relatively subtle health inequalities in youth (West 1988; West, Macintyre et al. 1990; West 1997; West and Sweeting 2004; Hagquist 2007; Hanson and Chen 2007), SEP differences in health seem to emerge rapidly when heading into adulthood (Rahkonen, Arber et al. 1995; Pensola 2004). One possible



explanation for the rapid emergence of health differences in early adulthood is that many behavioural and environmental determinants of health and health differences are established at this phase of life. Many lifestyle factors and behaviours that are often adopted during youth (such as smoking, alcohol abuse and physical inactivity) have substantial long-term consequences for young people's health (Hurrelmann 1990), which may become apparent when heading into adulthood. For example, changes in dependence (Furlong and Cartmel 2007) and transitions into adulthood (Coles 1995) may form the lifestyles and behavioural patterns that are relevant to the emergence of health differences. Separation from childhood family and childhood surroundings may entail a transition to financial independence and to new living conditions which affect the individual's health behaviour and health. However, it is possible that the effects of early environment and life-course do not become apparent until this stage of life, as suggested previously (Rahkonen, Arber et al. 1995).

High SEP has been found in previous studies to promote better living conditions, healthier working conditions (Schrijvers, van de Mheen et al. 1998; Borg and Kristensen 2000; Monden 2005), as well as healthier lifestyles, attitudes and choices (Wardle and Steptoe 2003). Furthermore, it has been shown to be associated with physically less strenuous and psychosocially more rewarding work and better housing conditions than lower SEP. All these factors may lie behind the educational differences seen in the health of young adults. Moreover, those with high SEP have been found to have less detrimental health behaviours (Sobal and Stunkard 1989; Droomers, Schrijvers et al. 1999; Lindström, Hanson et al. 2001; Martinez-Gonzalez, Varo et al. 2001; Casswell, Pledger et al. 2003; Paavola, Vartiainen et al. 2004; Laaksonen, Rahkonen et al. 2005; Power, Graham et al. 2005; Ali and Lindström 2006; Roos, Talala et al. 2008). However, health behaviour in youth and health itself can have an influence on SEP. Based on selection mechanisms, those with poorer health (Haas 2006) and a health-damaging lifestyle and behaviours (Koivusilta, Rimpelä et al. 1998; Koivusilta, Rimpelä et al. 2001) may end up in lower educational tracks, unfavourable employment paths and low SEP in adulthood. Some studies have even suggested that the relationship between adult SEP and health outcomes is mainly attributable to selection effects rather than a causal effect of SEP exposures on health and behaviour (Osler, McGue et al. 2007). Furthermore, differences in health by SEP may arise from circumstances and experiences in early life which affect one's education, living conditions and health behaviour and further, health.

#### Childhood circumstances

According to the findings of this study, childhood circumstances contributed to the educational health differences seen in poor SRH. In general, those suffering from an unpredictable home life in childhood seemed to have an increased risk for poor

health and lower education in early adulthood. Previous studies have also reported that childhood circumstances are associated with one's educational and employment pathways (Pensola 2004) as well as indicators of health (Lundberg 1991; Rahkonen, Lahelma et al. 1997; Dube, Felitti et al. 2003). Childhood circumstances explained a substantial part of the socioeconomic health differences in poor SRH in this study. Taken together, the childhood circumstances included in the analyses explained one-quarter of the differences between the highest and the lowest educational groups in poor SRH. Earlier studies have also found that this relationship between adult SEP and health is influenced by childhood circumstances. In some cases the associations have been somewhat weaker than in this study, but the general direction of the findings has been the same (Lundberg 1991; Power 1991; van de Mheen, Stronks et al. 1998). A study based on the British 1958 birth cohort suggested that SEP differences in health at age 23 were not eliminated after taking account of earlier circumstances, but the differences were substantially reduced by adjusting for a number of factors in childhood, in particular SEP, housing tenure, crowding, family size and receipt of free school meals (Power 1991).

In this study the strongest childhood factors contributing to educational health differences were parental divorce and in men, living in a single-parent family in childhood. Corresponding findings for the role of the single-parent family have been reported previously regarding mortality (Pensola 2004). Other childhood circumstances contributed to educational health differences as well. In particular, having been bullied at school was a strong determinant, which may be associated with later trajectories due to psychological mechanisms.

### Health behaviour

Health behaviours are important determinants of health and health inequalities (Townsend and Davidson 1982; Stronks, van de Mheen et al. 1996). Health behaviour explained almost half of the health differences between the lowest and the highest educational category in young adults in this thesis, which is consistent with findings concerning broader adult age groups (Lynch, Kaplan et al. 1997; Barger 2006; Laaksonen, Talala et al. 2008). Some studies, however, have suggested that the role of health-damaging behaviours in lower SEP groups as a mediating mechanism is small (Lantz, Lynch et al. 2001), or at least smaller than the effect of material factors (Schrijvers, Stronks et al. 1999). In this study, daily smoking made the largest contribution to educational health differences, but heavy drinking, nutrition (as indicated by the use of vegetables), obesity and physical activity also played important roles. It is notable that behavioural patterns are partly adopted before the final level of education has been determined and, if behavioural patterns acquired early in life affect the later educational track as has been suggested (Koivusilta, Rimpelä et al.

2003), behavioural patterns adopted at young age may partly cause educational health differences. However, it is also evident that low level of education increases the risk of many health endangering behaviours, and behavioural factors may thus partly mediate the effect of education on health.

The prominent role of daily smoking is not surprising as smoking is one of the most constant health behaviours from adolescence to adulthood (Paavola, Vartiainen et al. 2004). Previous research confirms that smoking is indeed an important contributing factor to health inequalities. Power and colleagues found in the 1990 that smoking habits at 16 years of age explained a substantial part of the variation in self-rated health in young adults (Power, Manor et al. 1990). Heavy alcohol use, which in this thesis substantially reduced the educational differences in poor SRH, has been identified as an important factor in previous studies as well (Casswell, Pledger et al. 2003). Although the use of vegetables is an adequate proxy for a healthy diet (Roos, Talala et al. 2008), it is still only one part of healthy nutrition. However, a recent Finnish study showed that vegetable use contributed strongly to educational differences in both cardiovascular and total mortality in adults (Laaksonen, Talala et al. 2008). The impact of obesity on health inequalities probably increases with advancing age.

#### Current circumstances

Living conditions in early adulthood may in some cases precede both the level of education and health, and influence their development, but the opposite causal order may be more important: living conditions in early adulthood are likely to be partly determined by the level of education. In this study, current circumstances explained almost one fifth of the differences in poor SRH between the highest and the lowest educational category, and this was completely due to the effect of main activity, as low education and poor health were particularly common in unemployed respondents. This clearly underscores the role of employment paths in the development of health inequalities. Earlier studies have also reported poor health and low education among unemployed young adults (Berth, Forster et al. 2003; Ahs and Westerling 2006). The effect of current circumstances on health differences has been identified in other studies as well. In a study based on the 1958 British birth cohort, recent experiences of unemployment and family formation were found to have an important role in explaining educational health differences (Power 1991). Unemployment is a particularly significant phenomenon in early adulthood, as exclusion from the wage labour system may have serious consequences for later life and adjustment to society. As was shown in this study, the pathway from adverse childhood experiences to education-based poor health is partly due to the fact that those from adverse circumstances are more likely to face unemployment, which affects health, either directly or potentially through health behaviours.

## Mechanisms behind the health differences

Childhood disadvantage and adverse circumstances affect health in adulthood through various processes, for example the child's development, health behaviours and the associated educational, work-related and social pathways (Graham and Power 2004). According to the findings of this thesis, the effect of childhood circumstances on the differences in poor SRH between the lowest and the highest educational category was largely shared with the effect of behavioural factors adopted by early adulthood. Almost two-thirds of the effect of childhood circumstances was shared with behavioural factors, daily smoking being the single strongest factor. Almost one-fifth of the effect was shared with the respondent's current circumstances, mainly with the employment path.

The findings of this thesis can be interpreted to support the role of both material and behavioural mechanisms in the development of health inequalities. Education may affect later living conditions and health behaviour, which in turn influences health, and the findings are consistent with this pathway. On the other hand, it is not possible to rule out the hypothesis that health-related selection significantly contributes to the observed socioeconomic health inequalities (Haas 2006), as it was not possible to comprehensively measure health in childhood and adolescence. The respondents' retrospective self-reports on their own chronic or long-term illness, however, did not explain the educational differences in health at all ( $< 1\%$ ), and self-reported childhood morbidity was not associated with education. This finding suggests that health-related selection does not explain health inequalities in young adults in Finland. Other selection mechanisms, however, may have a more important role. Early initiation of smoking or heavy drinking, for example, may have affected both educational pathways, as suggested in previous studies (Koivusilta, Rimpelä et al. 1998; Koivusilta, Rimpelä et al. 1999), and health, but it was not possible to analyse the importance of these pathways in this study.

The findings suggest that socioeconomic health differences are partly due to the fact that early social circumstances affect both educational achievement and health in adulthood. This is often described as "indirect selection", which refers to a situation where low SEP does not in itself cause poor health, but where low SEP and poor health are both caused by a third factor. The findings point at a pathway from childhood social circumstances to adult educational health differences through an uneven distribution of health behaviours and unequal adult circumstances, mainly differences in employment. However, it seems that childhood social circumstances affect educational health differences through other mechanisms as well, but these remain unidentified in this study. The role of psychosocial and work-related factors in explaining the remaining health differences have to be considered (Schrijvers, van de Mheen et al. 1998; Borg and Kristensen 2000; Monden 2005; Karmakar and Breslin

2008). Regarding working conditions, it has been suggested that a large part of the social class differences in physical as well as mental illness is a result of systematic differences between classes in living conditions, primarily differences in working conditions (Lundberg 1991). In this study, however, only part of the young adults were currently employed full-time or part-time. Lifetime exposure to adverse working conditions may explain a significant part of the health differences. The qualitative aspects of work also have a bearing on health inequalities. For example, the relationship between SEP and health can largely be attributed to job control whereas job demands reinforce the relation (Rahkonen, Laaksonen et al. 2006). Some results suggest that stressors may be an important mechanism underlying the social gradient in health (Orpana, Lemyre et al. 2007), although they affect health differences together with other factors (Lundberg 1997).

### 7.1.5 Gender differences

This thesis found that childhood determinants of health and health behaviour differed between men and women. However, gender differences were found even in reporting childhood adversities. A clearly larger proportion of women than men reported childhood adversities. The difference was particularly marked in the case of items open to interpretation (e.g. conflicts within the family). It can be suggested that girls may be more sensitive to these problems in childhood and also be more prone to report them. Many studies have found that women report higher rates of morbidity, disability and health care use than men do (Lahelma 1993; Adler, Boyce et al. 1994; Manderbacka, Lundberg et al. 1999), although there are also studies that indicate no clear gender differences (Cohen, Forbes et al. 1995; Macintyre, Ford et al. 1999). In this study, women reported psychological distress and somatic disorders more often than men did. However, men rated their health poorer than women. On the basis of these data it is not possible to assess the extent to which these gender differences in self-reported health reflect gender patterns in reporting and to what extent they arise from gender differences in different dimensions of health.

Women seemed to be more susceptible to childhood circumstances that determine current daily smoking. Corresponding findings have been reported earlier (Jefferis, Power et al. 2004). The mother's role was particularly significant for women: mother's alcohol and mental health problems determined women's daily smoking in the age-adjusted model. This study replicated the earlier finding concerning the effect of maternal smoking on girls' smoking (Kandel, Wu et al. 1994; Kandel 1995). Health behaviour traits in offspring may depend more on mother's than on father's health behaviour. Both differences and similarities were found in the childhood determinants of heavy drinking in adulthood. In both men and women heavy drinking was affected by childhood circumstances. However, the childhood predictors of heavy drinking

were largely different in men and women. More research is needed to explore the gender differences in the life-course determinants of drinking. Earlier research has revealed systematic gender differences in drinking patterns and determinants, and biological, social and cultural reasons have been suggested as explanations for these differences (Holman, Jensen et al. 1993; Lo 1996; Ahlström, Bloomfield et al. 2001; Kuntsche, Gmel et al. 2006). However, some studies have reported only moderate gender differences in adolescence (Bahr, Marcos et al. 1995).

Overweight was quite weakly associated with childhood circumstances. In women, childhood circumstances (low parental education, parental unemployment and single-parent family) were associated with overweight, but the effects were largely attenuated by other childhood circumstances. In addition, the effect of parental education was partly mediated by the respondent's own educational level and other living conditions. In men, no significant childhood predictors were found for overweight. The lack of strong associations may be due to the characteristics of the cut point a BMI of 25 does not accurately distinguish overweight from normal weight men (Garn, Leonard et al. 1986). The effect of childhood may be more apparent when BMI is higher. Overweight young adults are likely to be a heterogeneous group, whereas obese persons constitute a more distinct group with specific determinants.

The effects of childhood circumstances on health may be gender-specific. Earlier studies have reported marked gender differences in the associations between reported childhood experiences and environmental circumstances and adulthood mental disorders, for instance. In women, a greater number of childhood adversities were found to be associated with mental disorders in adulthood, and the statistical significance of these associations statistical significance was greater than in men (Pirkola, Isometsä et al. 2005). According to the results of this study, it seems that health in men and in women is predicted by partly the same and partly different childhood characteristics. It has been suggested that women are more vulnerable to the effects of adverse childhood experiences than men. On the basis of this research it is not possible to disprove this hypothesis, even though the determinants seem to be different. More research is needed to explore gender differences in childhood predictors of health and health behaviour.

## **7.2 Methodological considerations**

This study was based on a nationally representative sample of young Finnish adults. Its main strengths lie in the breadth of indicators chosen to describe childhood circumstances and various adversities. Another important strength is that it has been possible to study them jointly with several adult characteristics. However, due to the limited study population, it was possible to report only strong associations. The cross-sectional and retrospective nature of the data also raises some methodological issues. In addition, the validity of the measures used should be carefully assessed.

## 7.2.1 The cross-sectional and retrospective nature of the data

One obvious limitation of the present study is the cross-sectional and retrospective nature of the data. For instance, it is only possible to approximate the age at which the respondent had been exposed to adversities during childhood. The possible effects of parental divorce, for instance, on later health and educational achievement may depend on the age at which this adversity is experienced. However, no moderating effects of age were seen in the association that was found between childhood parental divorce and adult psychological distress in a British national birth cohort at ages 23 and 33 (Rodgers, Power et al. 1997). A corresponding finding has been reported regarding the association between parental divorce and subsequent well-being (Sigle-Rushton, Hobcraft et al. 2005).

Retrospective information on childhood conditions may also give rise to another kind of bias. It is possible that current health or its determinants to some extent affect retrospective perceptions of childhood conditions and problems ('negative affectivity'). As has been suggested previously, results based on retrospective reports should be interpreted with caution (O'Malley, Carey et al. 1986; Dube, Williamson et al. 2004; Hardt and Rutter 2004). In addition, it is possible that people use different criteria when reporting childhood problems. This is a problem if the criteria vary systematically according to the other variables used in this study. However, there is no reason to suspect that that is the case. Underreporting is another possibility when recalling childhood experiences. However, a methodological evaluation of a study exploring the association between childhood maltreatment and adult depressive disorders concluded that even if there was some underreporting of both early adverse experiences and adult depressive episodes, that was unlikely to threaten the conclusions drawn about the link between them (Brown, Craig et al. 2007). It has been suggested that as many life-course data are collected retrospectively, it would be valuable to know how items of information are recalled with what degree of accuracy and over how many decades. It would be interesting to know which methods of collecting these retrospective data maximise accuracy (Blane, Netuveli et al. 2007). However, no comprehensive picture of this is available.

Studies of social mobility based on longitudinal or register-based data provide strong evidence on the association between primary and later economic circumstances. However, the research evidence indicates that this association seems to be much weaker when the economic situation of the childhood home is assessed retrospectively. There are at least four potential sources of inaccuracy: first, children very rarely know the family's economic situation; second, children do not know to what they should be comparing their family's situation; third, economic situations have been different in different decades; and fourth, there is some mobility even in the respondents' economic situation. It seems that the absence of points of comparison for the respondent in these assessments causes bias that cannot be corrected by age-adjustment (Moisio and



Karvonen 2007). In this thesis, however, assessments were provided by a rather narrow age group, which improves their validity to some extent as the respondents have spent their childhood in rather similar circumstances. In addition, the childhood adversities studied in this thesis described more social than economic aspects of childhood.

Although cross-sectional data do not offer the same benefits as a longitudinal data setting, there are good reasons to argue that the cross-sectional design with retrospective inquiries can yield reliable information. First, earlier studies have reported good test-retest reliability for reports on childhood, at least in the ACE Study in the United States (Dube, Williamson et al. 2004). Second, the information on living conditions and on health status/health behaviour was collected as part of a major survey not specifically focused on the data considered here. The life-course perspective as a study design has many benefits in a longitudinal setting, but it also involves potential problems, most notably the very long time required for data collection, the fixed sample structure and the risk over time of increased sample loss (Wadsworth, Butterworth et al. 2003).

In this study it was not possible to take into account the impact of prenatal conditions. However, environmental and biological factors certainly play an important role in determining health and health behaviour. A life-course approach offers a way to conceptualise how underlying socio-environmental determinants of health, experienced at different life-course stages, can differentially influence the development of chronic diseases, as mediated via proximal specific biological processes (Lynch and Smith 2005). Although this was not possible in the present study, it would be useful to incorporate measured aspects of the environment into genetically informative twin models to begin to understand how specific environments and phenotypes are related to various health outcomes (Dick, Pagan et al. 2007). There is a growing recognition that the risk of many diseases in later life is affected by adult as well as early-life variables, including those operating prior to conception and during the prenatal period. Most of these risk factors are correlated because of common biologic and/or social pathways, while some are intrinsically ordered over time (De Stavola, Nitsch et al. 2006). The life-course approach has been recommended especially in studies on substance abuse, as it may integrate biological, psychological and social causes of drinking in the same models (Andersen 2004).

Could the effects of childhood or health behaviour on health be different in different educational groups? Although this question was not in the focus of this study, it is one that warrants serious consideration. A Norwegian study assessed the effect of education and lifestyle factors on people's self-reported health and contrasted two models in explaining health inequalities: the mediation model and the moderator model, hypothesising that the effects of the lifestyle variables on health could be dependent on educational level. In this study, both education and lifestyle factors had expected effects on health, but the results did not permit a conclusion as to which one of the two models of educational-related health inequalities should be preferred (Thrane 2006).



### 7.2.2 Non-participation

A high proportion of non-participants will undermine the reliability of the results if the characteristics of this group differ significantly from those of the participants. In this case the results on the prevalence or level of the phenomena studied will not necessarily provide a true picture of the situation in the study population and the results cannot be generalised to that population. One of the main aims of the Health 2000 Survey was to produce as reliable assessments as possible of the prevalence of various health problems and associated factors in the adult population in Finland. Every possible effort was made to collect data from as many people in the survey sample as possible, and indeed the participation rate was high throughout the Health 2000 Survey. This thesis used this large nationwide dataset in order to obtain generalisable results. The sampling design was developed by Statistics Finland based on two-stage stratified cluster sampling. In addition, a specific weighting system was constructed for data analyses to minimise the effect of errors due to the sampling design and non-participation. The purpose of these weights was to enable to correct the sociodemographic distribution of the respondents to correspond with that in the sample. The weights were calibrated according to poststratification defined by age, gender, area and language. This increases the reliability of the results considerably. In the Health 2000 Survey the participation rate was unusually high. Non-participation analyses have been conducted for the Health 2000 Survey (Laiho and Nieminen 2004; Laiho 2006; Koskinen, Laiho et al. 2008)

Participation rates were much higher among young women than men aged 18–29 years: 7% of women and 12% of men did not take part in any stage of the survey. In women, differences between age groups were marginal, whereas in men non-participation was highest in the age group 25–29 years. Participation rates were roughly the same in Finnish and Swedish-speakers, but more than one-quarter of those with some other mother tongue did not take part in any stage of the survey. Participation was lowest in the regions surrounding Helsinki and Tampere. In general, non-participation was slightly higher in urban than in rural areas (Koskinen, Laiho et al. 2008).

By socioeconomic status, the participation rate was lowest in the small group of pensioners and in the categories of “others” and “unknown”. Participation in upper-level employees and students was slightly above average. Non-participation was clearly higher among unemployed men than in those who were employed, regardless of the duration of unemployment. In women short-term unemployment did not increase non-participation, but in women who had been out of work for more than 6 of the 12 months preceding the survey, non-participation was high. In both women and men non-participation was highest in those with the lowest level of education and lowest in the group with an academic degree. The participation rate was slightly lower than average in the lowest income brackets, but otherwise the differences between income groups

were quite marginal. In women, differences by marital status were minor, whereas in men those who were married or co-habiting were the most active participants. Men who lived alone did not take part in the survey quite as actively as those who lived in larger household-dwelling units, but otherwise the size of household-dwelling unit did not show a strong correlation with participation rate (Koskinen, Laiho et al. 2008)

These findings give no reason to suspect that variation in the participation rate has decisively affected the results of this thesis, bearing in mind that the weighting system developed for the statistical analyses corrects part of the errors. However, it is noteworthy that especially in the case of heavy alcohol use and mental disorders, and possibly in the case of smoking and poor health, the data may exclude many of those with serious problems. Thus, the prevalence of such problems examined in this study may be an underestimation.

### 7.2.3 Methodological considerations of the measures used

A few points must first of all be raised concerning the reliability and validity of the outcome measures used in this thesis. In general, it has been suggested that social scientists need to be more critical of such measures as self-rated health in life-course studies since they lack an aetiology and, thus, biological plausibility (Blane, Netuveli et al. 2007). However, SRH was chosen as the main measure of health in this thesis because there is strong evidence that it is a particularly good measure especially in early adulthood when clinical endpoints are uncommon (Power, Matthews et al. 1998), and because it is a good and valid measure of health (Krause and Jay 1994; Martikainen, Aromaa et al. 1999). In addition, several studies have shown that it has strong predictive value of further health. SRH is a very strong predictor of functional capacity (Idler and Kasl 1995; Ferraro, Farmer et al. 1997), other future health problems (Kaplan, Goldberg et al. 1996; Moller, Kristensen et al. 1996) as well as mortality (Mossey and Shapiro 1982; Kaplan and Camacho 1983; Idler and Benyamini 1997; Mackenbach, Simon et al. 2002; Martikainen, Aromaa et al. 2002). As for educational differences in SRH, one possible source of bias in the results is that different social groups may report their health differently. However, there is no strong evidence to support that.

The psychological well-being of young adults was assessed using the 12-item version of the General Health Questionnaire (GHQ12). This is a self-report questionnaire designed for the assessment of current mental state, and it has been found to be a valid and reliable measure of mental health (Goldberg 1972; Goldberg, Gater et al. 1997; Martin 1999; Pevalin 2000). The GHQ remains the gold standard in measuring psychological distress. It has been found to predict future morbidity and premature mortality in the adult population (Robinson, McBeth et al. 2004; Nicholson, Fuhrer et al. 2005). GHQ was originally designed as a screening instrument and in fact does not

distinguish between depressive and anxiety disorders. In this thesis, the commonly used cut-off point of 2/3 was applied to define those with psychological distress. The choice of the GHQ method was motivated by the fact that it gives a rather good estimate of the individual's current mental state and because the purpose was to define those with psychological distress, without any need to distinguish between depressive and anxiety disorders.

The measure of somatic morbidity used in this thesis was based on several questions inquiring whether a doctor had ever diagnosed the respondent as having a specified chronic somatic disease. In addition, a complementary open-ended question coded on the basis of the ICD classification was used. The measure of somatic diseases was thus specifically constructed for this research. However, a similar approach has been successfully used in many earlier Finnish national health surveys. Comparisons with simultaneous clinical examinations have shown (Heliövaara, Aromaa et al. 1993) that the agreement between open-ended self-reports and doctors' diagnoses depends on the condition and ranges from excellent (cardiovascular diseases) to moderate (musculoskeletal diseases). In this research the respondents were considered to have a somatic disease if they reported at least one of those in the list of 33 somatic disorders, ranging from serious congenital conditions to milder chronic somatic disorders. For some diseases additional criteria were set. For example, asthma, arrhythmias, hypertension, back disorders, allergic and skin diseases as well urinary infections were only considered to be present if the respondents reported being in a physician's care or using regular medication because of their disease. This made it possible to exclude milder somatic symptoms from more serious ones. However, the disease spectrum naturally represents somatic disorders typical of young adults as described in Chapter 5.2.1.

The measure of daily smoking was based on self-report. Self-reported smoking has been found to be a valid measure in previous studies (Patrick, Cheadle et al. 1994; Rebagliato 2002), and the smoking index used in this study has been successfully applied in other Finnish studies as well (Helakorpi, Prättälä et al. 2008). The validity of the measure in this thesis was probably enhanced by the fact that the determinants of daily smoking, and not occasional or former smoking, were examined. In other words, the results refer to more or less persistent smoking which has pathways to future smoking and health as well. It is possible that the initiation of smoking is impacted by rather similar factors as the maintenance of smoking. However, this could not be reliably established in this study.

The outcome measure for unhealthy alcohol use was heavy drinking. In general, self-reported alcohol consumption tends to underestimate true consumption (Poikolainen 1985), the reasons varying from recall bias to feelings of shame and guilt over large amounts of alcohol. However, there are some other concerns regarding the measure

of heavy drinking. First, some detrimental health consequences of alcohol use grow linearly without any specific threshold; second, as mentioned above, respondents tend to underestimate their drinking; and third, the comparison group includes not only abstainers and moderate drinkers, but also ex-drinkers, who may have quit drinking due to health-related problems. Furthermore, the risk limit of total consumption is no guarantee for health: heavy episodic drinking may have severe health consequences even if total consumption does not surpass the limit (Salaspuro, Alho et al. 2005). Heavy episodic drinking would have been a relevant indicator of drinking problems in young adults: some studies have even suggested that the frequency of drunkenness may be the single best indicator of problem drinking in this age group (Bailey 1999). However, there was no opportunity to use a good measure of that. The accuracy and validity of self-reported alcohol use have frequently been discussed (Del Boca and Darkes 2003). However, there is no reason to expect that childhood circumstances would markedly affect bias in self-reported drinking. It is also worth noting that the survey data possibly exclude a major part of those with severe substance use problems.

BMI is an accepted measure of obesity in population studies. It is an indirect measure of body fatness, but provides more reliable results than some other ratios of weight to height. The only exceptions to the reliability of BMI are persons of extreme age, very muscular build and extreme height (James 2004). In population studies, weight and height are often assessed by self-report. It has been suggested that people tend to underreport their weight and overreport their height, which leads to an underestimation of BMI. Therefore, the accuracy of self-reported measures have frequently been questioned (Bolton-Smith, Woodward et al. 2000; Engström, Paterson et al. 2003). However, there is no reason to expect that childhood circumstances would markedly affect bias in self-reported weight and height.

Methodological aspects of the measures of childhood circumstances and adversities were already discussed earlier in Chapter 7.2.1. It is notable that early adulthood is a phase of life when living conditions often are not yet stable. There is no reason to expect that people would tend to misreport neutral living conditions such as education, main activity and family structure. Education probably was the most influential factor in this thesis and the measure of it was carefully constructed, taking into account expected and achieved levels of education. The measure of the respondent's own educational level could not take into account the fact that some subjects possibly had completed their education only temporarily – and yet in the case of detrimental health behaviours, for example, those who continue their education later may come to resemble those who already have a higher level of education. It is therefore possible that the effect of own education would have been even stronger if it had been possible to anticipate the final level of education of all participants. As regards current family

structure, it is notable that part of the respondents still lived with their parents. The inconsistent results for the effect of family structure on health and health behaviour are probably due to fact that the process of family formation in these young age groups is still underway and is strongly dependent on age as well. However, family structure or having children were not used as main measures in this thesis.

### **7.3 Implications for future research**

This study improved our understanding of the life-course determinants of health, health behaviour and health inequalities in young Finnish adults. However, it is clear that we will need to understand the causal mechanisms involved in even greater depth if we are to develop successful interventions. Therefore, future research should include both longitudinal observational studies and intervention studies to find the best ways of preventing adversities that result in ill-health at as early a stage as possible. It would also be useful to examine causal hypotheses in relation to several health determinants and health outcomes. In observational cohort settings the number of observations point should be increased (and their intervals decreased). In particular, it would be important to have more detailed knowledge about the age at which certain childhood adversities were experienced and how they were associated longitudinally with later adversities and other health determinants. Important advances could also be made by using both repeated survey data and data drawn from registers.

One of the main findings of this study was that those young adults with only primary level education were at the highest risk of poor health and health-damaging behaviours. Future research should use relevant data to assess whether these young adults are at high risk of social exclusion and how the problems are accumulated at this age, and furthermore what are the most important life-course predictors of social exclusion. In particular, it is necessary to assess the accumulation of health-damaging behaviours with unfavourable circumstances.

Finally, while it is obviously important to identify the causal mechanisms at play, research should also strive to promote health and prevent illness. Therefore, opportunities for early interventions should also be tested. This means that some of the long-term repeated follow-up studies include efforts to develop possible intervention methods. In view of the nature of present childhood adversities and their consequences, interventions should probably be carried out with families and schools.

## 7.4 Implications for social and health policy

Public health has improved considerably in Finland over the past few decades. However, the continuity of this trend is under serious threat. A few main points deserve separate mention here. First of all, the population is ageing very rapidly, which presents a whole set of new major challenges to the health and social sector. Secondly, socio-economic health inequalities have continued to persist or even increased – even when measured in absolute terms. Thirdly, it seems that the incidence of several health problems is now on the increase. There is evidence of adverse trends in important behavioural and environmental factors (e.g. substance abuse, overweight and increasing stress in the workplace) that are increasing the risk of many public health problems and the growth of ill health. Many of these alarming trends are accentuated in the young adult population (Koskinen, Kestilä et al. 2005).

The Finnish Government's policy programme for *Children, Youth and Families* (2007–2011) underscores the importance of investing in the well-being of children, young people and families. This emphasis seems well-placed in view of the results of this thesis. The programme suggests that the risks associated with growth and development are identifiable in early childhood, and makes clear that the resources to ensure the secure development of children and the youth, and the well-being of families will be made available in various sectors. The foundations for health are laid in childhood and youth, as is confirmed by the results of this thesis. Childhood circumstances are important in determining health, health behaviour and health inequalities in early adulthood. The early recognition of childhood adversities followed by relevant support measures may play an important role in preventing health problems in adulthood. Understanding the reasons and pathways to poor health and health inequalities and improving the living conditions of families with children, could prevent the unfortunate pathways that lead to the development of poor health, health-damaging behaviour and health differences. It is important to recognise the needs of children living in adverse circumstances and pay attention to reduce parental smoking and heavy drinking. Single-parent families in particular would benefit from support. Even without a complete understanding of the pathways involved, research results support the assertion that improving the living conditions of children (Reynolds, Temple et al. 2001) would have long-term benefits for adult health and may be an especially powerful means towards the reduction of health inequalities. However, despite the general acceptance of the need to help children suffering from adversities and health risks, it is no simple task to provide the necessary know-how and resources. The early intervention into problems related to family life and the first years at school should be an everyday practice. This underlines the need to develop effective approaches for such interventions and to demonstrate that they actually lead to health improvements.

At the same time as the overall level of public health level has continued to rise in Finland as well as in many other European countries, health disparities between subgroups of the population have persisted or even increased (Mackenbach, Kunst et al. 1997; Lahelma and Koskinen 2002; Mackenbach, Bos et al. 2003; Palosuo, Koskinen et al. 2007; Valkonen, Martikainen et al. 2007). As pointed out in a recent report of a WHO collaborative cross-national study on health inequalities among young people in the WHO European Region and North America, the health and well-being of many young people give cause for celebration, yet sizeable minorities are experiencing worrying problems related to obesity, self-esteem, life satisfaction, substance misuse and bullying (Currie, Gabhain et al. 2008). It seems that inequalities start to increase when heading into adulthood as early adulthood is very often a period of adopting persistent health behaviours as well as living conditions. The reduction of health inequalities has been an important public health goal in Finland and elsewhere for years (WHO 1986; Ministry of Social Affairs and Health 2001). In recent years special effort has been invested in research on health inequalities as well as in developing policies and interventions to reduce them, as exemplified by *the National Action Plan to Reduce Health Inequalities in Finland 2008-2011* (Ministry of Social Affairs and Health 2008), which ties in closely with *the Government Policy Programme for Health Promotion* (2007–2011). One main goal of this program is to improve the general state of health of the population and to narrow the health gaps between individuals. This thesis contributes to the specific questions of health inequalities and the mechanisms by which health and health inequalities are determined. In particular, it underlines that health inequalities develop in the life-course and emphasises the role of childhood circumstances in this process. In order to reduce health inequalities at the population level, it is essential to take account of the whole life-course. This thesis provides a useful background for developing health promoting habits, environments and interventions to reduce the risk of ill-health and health inequalities in today's young adults and tomorrow's middle-aged and elderly population.

National social and health policy as well as health promotion are faced with a number of challenges. Young adults who have no more than primary education are at greatest risk of poor health. A low educational level and unemployment are closely associated with health-damaging behaviours and poor health, a point that must be emphasised in planning preventive strategies. Those outside the educational system and employment are in a worse situation regarding many health behaviours and living conditions than their higher-educated peers (Koskinen, Kestilä et al. 2005). It is of paramount importance to ensure a secure transition from school to further education and employment. In addition, it is crucial to recognise the needs of those young people outside the schooling system and working life, to prevent unfortunate trajectories to later life and the possibility of accumulation of problems as well as the risk of social exclusion. Poor health can be considered to have a role in the social exclusion,



and reducing health inequalities is one way to prevent social exclusion (Ministry of Social Affairs and Health 2008). The Finnish Youth Act also emphasises the need for policies to support the empowerment of young people with a view to preventing social exclusion and improving the living conditions of young people by adequate youth policy.

Smoking and heavy drinking are very important contributors to educational health differences even in early adulthood. These habits are often adopted in adolescence and it is therefore essential to have preventive policies to tackle early initiation. School and community-based smoking prevention programmes have been found to be particularly effective in preventing smoking onset among adolescents (Vartiainen, Pennanen et al. 2007). Also, as has been suggested before, programmes that are aimed at reducing smoking among socially disadvantaged adolescents would have the effect of reducing smoking and smoking inequalities in adults (Novak, Ahlgren et al. 2007). Smoking and heavy drinking among young adults should also be specially targeted. Interventions should aim to prevent addiction to smoking among the lower educated by means of pricing policies, school-based programmes, and smoking cessation support for young adults (Huisman, Kunst et al. 2005). More attention should be paid to heavy drinking in young adults and to factors that influence their drinking patterns (Delucchi, Matzger et al. 2008). As they are clearly predicted by childhood circumstances and parental substance abuse, preventive policies should give more attention to these factors as predictors of corresponding substance abuse in offspring.



## 8 CONCLUSIONS

This study examined the life-course determinants of health, health behaviour and health inequalities in young Finnish adults aged 18–29 years on the basis of the nationally representative Health 2000 Survey. The exploration of various retrospective measures of childhood circumstances and adversities alongside several measures of young adults' current circumstances helped to shed light, first, on the effect of different childhood circumstances on health, health behaviour and educational health differences later in life and second, on the pathways by which childhood and youth have an effect on them.

This study showed that childhood circumstances have an effect on the health and health behaviour of young Finnish adults. The impact of childhood on early adult health varies according to the measure of health: childhood conditions are strongly associated with poor SRH and psychological distress, whereas somatic diseases and disorders typical of young adults show no or only weak associations with these factors. These associations are relatively independent of later education.

Childhood circumstances seem to predict health-damaging behaviours, although current circumstances, and education in particular, partly or largely mediated the effects on the outcomes. These indicators of health behaviour (smoking, heavy alcohol use and obesity) are shaped throughout the life-course.

Another important conclusion of this thesis was that there are educational differences in health in early adulthood. After the hypothesised equalisation of youth, health differences seem to be marked even in early adulthood. Also educational differences in health behaviours exist. More importantly, this study indicated that childhood social circumstances have an impact on health differences in early adulthood, and this effect is largely shared with the effect of health behaviours adopted by early adulthood. This study lends further support to the assumption that health differences related to education also develop throughout the life-course.

## 9 ACKNOWLEDGEMENTS

This study was conducted at the National Public Health Institute's (KTL) Department of Health and Functional Capacity. It has been extremely rewarding for me to work in this highly professional, kind and supportive environment. I am deeply grateful for the excellent research facilities provided by KTL and the Department. In particular, I am indebted to the staff and participants of the Health 2000 Survey for high quality dataset I was able to use in this study. I gratefully acknowledge the financial support granted by the Yrjö Jahnsson Foundation (1.9.2003–31.8.2004), the Doctoral Programme for Population, Health and Living Conditions (1.9.2004–30.6.2008) and the Juho Vainio Foundation (1.11.2007–30.6.2008).

I could have not wished for more professional and more supportive supervision for my dissertation. Docent Seppo Koskinen, PhD Tuija Martelin and Docent Ossi Rahkonen, I feel deeply privileged to have had the opportunity to work with such wonderful people. Seppo Koskinen, my main supervisor, has been the strongest support for me. I sincerely admire his expertise in several fields of research, as well as his endless kindness, fairness and supportiveness. Tuija Martelin has contributed not only her friendship and excellent co-operation through the years, but her expertise in statistical methods has been of great value to me. Ossi Rahkonen has broadened my view on youth research and public health. I am constantly amazed at his patient, friendly and supportive encouragement and supervision. Thank you all.

I also express my sincere thanks to:

The reviewers of this study, Docent Leena Koivusilta and Docent Karri Silventoinen are gratefully acknowledged and appreciated for reading the manuscript and for their perceptive comments and constructive criticism that helped me to improve this summary. Thanks also to David Kivinen for his help with the English language.

The professionalism of my co-authors in the substudies has truly broadened my view on public health, psychology, statistics, sociology and epidemiology. I wish to thank Docent Hillevi Aro, Research Professor Arpo Aromaa, PhD Tommi Härkänen, PhD Kaisla Joutsenniemi, Docent Marjaana Lahti-Koski, Docent Kristiina Patja, PhD Tiina Pensola, Docent Sami Pirkola and Docent Kari Poikolainen for valuable advice and support. It has been a privilege to work with you.

The Doctoral Programme for Population, Health and Living Conditions (VTE) has provided me funding and support to conduct this PhD thesis. It has been a great pleasure to work in this programme. The doctoral VTE seminars were particularly helpful when I was conducting the substudies. Thanks to Tapani Valkonen, Pekka Martikainen, Ossi Rahkonen, Eero Lahelma, Ari Haukkala and Netta Mäki. I also wish to thank all the people who attended the seminars over these years. In addition,

thanks to CHESS doctoral students and supervisors for sharing seminars during these years. I have received both valuable comments and peer support that have helped to improve my work.

Riitta Nieminen, for the skilful layout of this thesis, Kari Piukka for creating the figures and Jussi Eskola, for the cover photo of this book.

The entire staff of our department, for creating such a pleasant atmosphere in which to work. Pirkko Alha, Virpi Killström, Noora Kuosmanen, Sirkka Rinne, Harri Rissanen and Ninni Vanhalakka for advice with various practical issues. Katri Kilpeläinen, Elisa Hyvönen and Mikko Pekkarinen for true friendship. Special thanks in KTL also to Tomi Mäkinen, Eila Linnanmäki, Taina Huurre, Sirkku Vuorma, Antti Hirvonen, Tarja Nieminen and Kirsi Talala; thank you for your support.

The Finnish Youth Research Network; thank you Mikko Salasuo, Tommi Hoikkala, Leena Suurpää and Anni Ojajarvi for your encouragement and friendship.

I warmly thank you all my dear friends for sharing these years with me; yes, there is life outside of research! You guys rock my world!

I reserve my deepest gratitude to my family for their love and support throughout these years. In particular, I wish to thank my dear father Matti and mother Riitta-Liisa and my kind and sweet sisters Elina, Leena and Ulpu. I could have not done this without you.

Helsinki, September 2008

*Laura Kestilä*

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## APPENDIX A

### *List of somatic disorders. Additional criteria in parantheses.*

1. Asthma (being treated by a doctor or currently using medication because of it)
2. Cardiac arrhythmia, rhythm disturbance (being treated by a doctor or currently using medication because of it)
3. Congenital heart defect or valve disorder
4. Other heart disease (for respondents over 30 years of age)
5. Stroke, other cerebrovascular disorder
6. High blood pressure (being treated by a doctor or currently using medication because of it)
7. Back disorder or other back defect (having been operated or being treated by a doctor or currently using medication because of it)
8. Rheumatoid arthritis
9. Other types of arthritis or rheumatoid conditions
10. Other musculoskeletal disorder
11. Visual defect or injury (being treated by a doctor or using other vision aid than glasses)
12. Hearing defect, hearing injury or disease causing impaired hearing (being treated by a doctor or using hearing aid or some other hearing help)
13. Diabetes
14. Disturbing allergy (being treated by a doctor or currently using medication because of it)
15. Chronic skin disease (being treated by a doctor or currently using medication because of it)
16. Crohn's disease
17. Celiac disease
18. Other inflammation of the large intestine (Colitis ulcerosa or other)
19. Other disease of the digestive system
20. Cancer

21. Bladder, urinary tract or a renal pelvis infection or bacteria in the urine (bothering repeatedly and being treated by a doctor or currently using medication because of it)
22. Recurrent migraine being treated by a doctor
23. Disorder of the kidney
24. Chronic infection
25. Intellectual disability
26. Other congenital disorder
27. Epilepsy
28. Gynaecological disease
29. Diseases of the blood and blood-forming organs
30. Respiratory disease
31. Disorder of the thyroid gland
32. Systemic connective tissue disorder
33. Other neurologic disorder